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REVIEW OF THE AVAILABLE LITERATURE ON THE LARYNX FOR 1960.*

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ANATOMY.

Kelemen and Sade[1] made an intensive study of the larynx of howling monkeys. All former investigators thoroughly agree that the noise produced by the howling monkey is the most intense produced by the larynx, far superior to the roar of lions. A detailed description of the larynx is presented. The sizes of the cartilages and air sacs are surprising, and the vocal muscles are massive. Kelemen and Sade state that because of the anatomic configuration it is utterly impossible to visualize the larynx with a laryngoscope. They theorize on the unusual volume of sound produced.

According to Semon's law, in partial laryngeal palsy, the abductor muscles are more susceptible to trauma than the adductor muscles. In an attempt to evaluate Semon's law Bowden and Scheuer[2] weighed the abductor and adductor muscles of the human larynx. The abductor muscles weighed only one-fourth as much as the adductor muscles. It is concluded that this inequality in bulk between the adductors

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and abductors might contribute to the apparent vulnerability of the latter and so provide some anatomic basis for Semon's law.

Kelemen[3] reminds us that the larynx is the source of sound but is not to be considered as the organ of speech. The larynx produces the raw material in the form of sound which is modified into voice by resonating chambers. To form speech, collaboration of the pharynx, tongue, palate, lips, and nasal cavity is necessary. This is a good article on basic anatomy and neurophysiology of speech.

Pressman, Simon and Monell[4] report the results of anatomic studies on dissemination of cancer of the larynx. They noted that cancer within the larynx spreads by direct extension and via the lymphatics. Their experiments were designed to study the anatomic patterns that might affect the spread by each of these routes. Observations dealing with direct spread were conducted by injecting dye into the larynges. Studies of the lymphatics were made by injection of dyes and radio-isotopes into living human, dog and pig larynges. Results and conclusions are well tabulated. All interested in this fascinating research problem should read this article.

PHYSIOLOGY.

Sonesson[5] made an anatomic and functional study of the human vocal folds and reported his observations in two parts. In Part I he studied the topographical arrangement of the muscle fibers in the vocal fold and the relation between the vocal muscle, the vocal ligament and the conus elasticus. He concluded that the vocal muscle is anatomically indivisible, arising from the thyroid cartilage and being inserted into the arytenoid cartilage with no fibers inserted into the vocal ligament. Posteriorly below the arytenoid cartilage and anteriorly close to the thyroid cartilage muscle fibers are inserted into the conus elasticus.

In Part II he describes a photo-electrical method for studying the vibratory movements of the vocal folds. The results of this study were summarized as follows: "*1.* The absolute

duration of the open period of the vibratory cycle diminished with rising pitch. *2.* The open quotient, *i.e.*, the ratio between the duration of the open period and the duration of the entire vibratory cycle, increased with rising pitch."

Sullivan and associates[6] made a study of the rotary component of the motion of the arytenoid cartilages in man. With use of a local anesthetic, direct laryngoscopy was performed by Sullivan with a movie camera focused at the posterior rima glottidis and the arytenoid cartilage. The developed film demonstrated a 45 degree rotary motion of each arytenoid combined with a lateral gliding motion.

Von Leden[7] presents his observations in a study of laryngeal physiology by means of ultra-slow motion pictures produced by high speed photography of 3,000 to 5,000 frames or pictures per second. These pictures give a new concept of abduction, arytenoid motion, and asynchrony of vibration. The films are only partially described.

Roentgenographic methods are being increasingly used to study the mechanisms and pathology of speech, according to Smith and associates.[8] The radiologist may, therefore, expect increasing requests for their services in speech investigation. Although progress has been made, many problems remain to be solved, for it is frankly admitted that the limits of normality are not as yet clearly defined.

Employing cinefluorographic studies on 10 normal subjects as they swallowed and as they phonated, Shelton, Bosma and Sheets[9] noted fairly consistent patterns of motion of the tongue, hyoid and larynx during the act of swallowing. Less consistent movements were noted in the various subjects on phonation, however. Shelton and associates believe that information from this research might be of use in devising a standard radiographic procedure for evaluating motor performance during strategic movements of the act of swallowing. Such studies as these do help to increase our understanding of normal function or of distortions incident to motor disability and/or to abnormal structural form. They

are, therefore, a definite contribution to the advancement of our knowledge of laryngology.

The technique of laminagraphic roentgenography is by no means a new medical diagnostic tool, but it has only limited use in laryngeal research. Hollien and Curtis[10] used this technique to investigate trends relating changes in the cross-sectional dimensions of the vocal folds with variations in vocal pitch. The study is well described and tabulated. Data showed significant group differences with low pitched subjects exhibiting larger vocal fold areas and thickness. Significant differences were also found between fundamental frequencies.

Investigative experiments by Rubin[11] in which motion pictures of the vocal cords were made with tracheotomized patients have convinced him of the validity of the aerodynamic theory of voice production. This subject, as well as the neurochronaxic theory, is well presented.

Barroilhet and associates[12] conducted experiments on a patient who had hemilaryngectomy, the larynx being left open so that the remaining vocal cord was readily visualized. With proper apparatus, vibrations of the cord and activity of the vocal muscle were simultaneously recorded. The conclusions were that in the absence of air currents the vocal cords do not vibrate and that no strict correlation can be made between the EMG of the vocal muscle and the vibrations of the vocal cords.

Guth and associates[13] conducted experiments in which the recurrent laryngeal and phrenic nerves were anastomosed on eight rats and three monkeys. Observations months afterwards led to the conclusion that the recurrent laryngeal nerve is capable of restoring function to the paralyzed hemidiaphragm in the rat and the monkey.

Brewer and Briess[14] prove the relationship between laryngeal disorders and industrial noise by an experiment on 12 patients. The patients were divided into four groups: controls, untreated noise exposed, treated noise exposed, and

treated unexposed. After exposure to noise, they showed hyperfunction of the thyroarytenoid muscle. The recovery time of the symptomatic patients, working daily in the presence of loud noise, was one hour as contrasted with ten to 30 seconds for controls and treated patients.

Advancing age is thought to be accompanied by progressive decrease in the acuity of perception and reflex activity. Adequate activity of the protective reflexes in the airway is of great importance, since depression or absence of such reflexes may lead to aspiration pneumonia or even death. Pontoppidan and Beecher[15] conducted studies to ascertain the response of the larynx evoked by mechanical or chemical stimulation. They showed that these protective reflexes are greatly decreased with aging. Results of the study also point to the hazard of further depression of the airway reflexes by the injudicious use of depressant drugs in the elderly.

A study was undertaken by Hollien[16] utilizing volunteers to determine the size differences between high pitched and low pitched voices. As a result of screening 254 volunteers, four groups of subjects were selected for study. A lateral X-ray procedure provided measurements of laryngeal dimensions. The results showed significant trends for laryngeal size to be smaller as pitch level becomes higher.

PATHOLOGY.

The pathologic effects of smoking on the larynx have been of interest to laryngologists for years. Little has been published on this subject. For this reason Devine's[17] comments are of particular interest. He tells us that it has been shown experimentally that tobacco is irritating to mucous membrane, stops ciliary activity, produces leukoplakia and contains carcinogens. Interesting clinical data are presented. Devine believes that leukoplakia and polypoid degeneration of the vocal cords are often associated with excessive smoking. Data clearly indicate that the risk of laryngeal cancer increases directly with the amount of tobacco smoked. Cancer of the larynx rarely develops in nonsmokers.

HISTOLOGY.

Perello and Comas[18] report results of exfoliative cytologic studies of the female larynx during the female genital cycle. They calculated the percentage of the eosinophilic cells and the cells with cario-pycnotic nucleus, which they found increases during ovulation and menstruation.

PHOTOGRAPHY.

Rubin and LeCover[19] describe a technique of high speed photography of the larynx, which differs from that used by the Bell Telephone Co. in 1940, in that they employ a camera with a movable laryngeal mirror, whereas the Bell camera had a fixed laryngeal mirror. The camera, light source and position of the patient are well described. It is concluded that this technique greatly enhanced the potentialities of high-speed photography in studying the behavior of the larynx in health and disease.

Peele[20] describes his method for photographing the human larynx step by step so that any one interested in this type of work could easily do it after reading this article. All of Peele's photography has been by indirect laryngoscopy in the office and his results are good. Peele states that laryngeal photography is unexcelled as a medium for studying the normal function of the larynx, for recording pathologic conditions of the larynx, and as a medium of teaching.

DIAGNOSIS.

Sheehan and associates[21] compared results of laminography in 60 patients with suspected neoplasms of the pharynx or larynx with those of direct and indirect laryngoscopy. The presence or absence of a lesion was demonstrated by both studies in 30 of the patients.

The laminogram failed to visualize the tumor in four of eight patients with benign lesions; however, in two patients the laminogram demonstrated undetected clinical laryngoceles. In 16 cases the laminogram demonstrated the inferior extension of the lesion, which could not be seen clinically.

Laminography resulted in one false positive diagnosis and three unsatisfactory studies. These results indicate that very early lesions cannot be detected and a differential diagnosis cannot be expected regarding inflammatory or neoplastic correlation.

It was found that the clinical and roentgenographic methods support each other and are indispensable in the correct differential diagnosis of a lesion in the pharynx and larynx.

Laryngography is a method of objective radiographic demonstration of laryngeal lesions, used by the radiologist to delineate masses within and about the larynx. Ogura and associates[22] are enthusiastic about this method, for it indicates accurate extension of the lesion and demonstrates impaired function of adjacent structures. The technique is well described. Successful interpretation of laryngograms depends upon the skill and speed of performance of the procedure and the experience and interest of the radiologist. No one can appreciate more than the surgeon, the importance of knowing the exact location and extent of the malignant lesion. According to Ogura and associates, correct interpretation of the laryngogram will, in most cases, supply this information.

Scalco, Shipman and Tabb[23] conceived the idea of using the Zeiss binocular microscope, employed in otitic surgery, for better visualization of laryngeal lesions when viewed through the Lynch suspension laryngoscope. This microscope is easily rearranged for uses with the suspension laryngoscope and, according to Scalco and coworkers, provides excellent visualization and magnification of the lesions on the cords. They claim that more precise, delicate surgical procedures can be performed because of the enlargement and brilliant visualization of the smallest lesion. The reviewers can testify that the late Dr. R. C. Lynch tried various types of magnifying glasses in an effort to obtain a better view of lesions on the vocal cords, with little or no success. His son, the late Dr. Mercer Lynch, used the Zeiss binocular operating microscope to view the larynx in conjunction with suspension laryngoscopy and also obtained photographs of

the cords through the microscope but never published his work. Scalco, Shipman and Tabb deserve credit for developing this new procedure.

Kovács[24] describes a method of asymmetric roentgenography of the vocal cords used in routine work on this organ. Diagrams and photographs are presented. This method is claimed to be particularly valuable for demonstrating subglottic extensions, as this area is usually beyond the visual range of the laryngoscope.

Direct laryngoscopy, after induction of general anesthesia, is advocated by Priest and Wesolowski,[25] who described the method employed by them for many years. A small endotracheal tube cuff is inserted through the larynx into the trachea, and it is inflated. This provides the anesthetist with a closed system for administration of the anesthetic. Priest and Wesolowski use a specially designed wasp-waisted laryngostat for exposure of the larynx and report highly successful results with their method. One of the reviewers has used a general anesthetic for direct laryngoscopy since 1922 and is in thorough accord with the views expressed in this article.

INSTRUMENTS.

Pope[26] experienced difficulty intubating a patient with a unilateral bony swelling of the upper right jaw; had a left handed laryngoscope been available, this difficulty would not have arisen. For this reason he devised such a laryngoscope for use in these cases.

Holinger[27] incorporated a significant modification to a previously described anterior commissure laryngoscope. Accentuating the principle of the Yankhauer post-nasal speculum, he flared out the proximal and distal portions of the instrument while leaving the center constricted. Holinger claims an easier and better view of the cords and anterior commissure is obtained.

Lewy[28] presents a new instrument to be used to obtain binocular vision through a tube. This instrument is attached to a headband. The optical system consists essentially of a

double mirror and prismatic system with an added optional slight magnification. With an optical viewing system involving both eyes, binocular vision is established with corresponding stereopsis and depth perception.

ANESTHESIA.

Yousef,[29] an anesthetist, describes the hazards of anesthetization in patients with postcricoid carcinoma. These lesions usually occur in middle-aged or elderly people, whose physical condition is usually below par. The surgical procedure is usually lengthy requiring prolonged anesthetization. The technique of induction of anesthesia for operation on these patients is described. Among 30 patients, two died during operation and another, three hours later.

Quevedo[30] calls attention to post-anesthetic traumatic granulomas, which are being seen with increasing frequency. He correctly states that these occur more frequently in women and usually at the vocal process; however, your reviewers recently saw a traumatic granuloma in the middle third of one vocal cord. Bilateral granulomas are also encountered. Quevedo advises waiting until the granuloma has become pedunculated before removing it surgically; however, while waiting for this to occur, he had the unusual experience of seeing a granuloma develop on the opposite vocal cord. It is interesting to note that, as a rule, in traumatic granulomas surgical removal is followed by complete cure. This is so different from contact ulcer granulomas that occasionally require repeated removals.

Conway, Miller and Sugden[31] conducted a study to determine the incidence and cause of sore throats after anesthetization. They were not able to determine the exact cause, but the incidence of sore throat in 1,259 patients after general anesthetization was 24.5 per cent. In 617 patients who did not have intubation the incidence was 10.2 per cent. Of 642 patients who had intubation, sore throat developed in 245 (38.2 per cent). The skill of the anesthetist, the type of anesthetic and the intubation tube used, all play an important part in the production of sore throat.

DISEASES.

Eighty-seven cases of laryngeal keratosis were followed by McGavran, Bauer and Ogura[32] from five to 15 years. Carcinoma of the larynx eventually developed in three patients. This does not support the prevalent concept that laryngeal keratosis or leukoplakia is a distinct precancerous condition. Conservative therapeutic measures and careful follow-up laryngeal examinations with biopsy as indicated, proved adequate in this series of cases. The warning is sounded that patients with atypia as well as keratosis have a higher incidence of persistence and should be observed more closely.

Many systemic diseases manifest themselves in the larynx. Maloney[33] discusses those most frequently encountered. He offers therapeutic suggestions for those problems arising from a hypometabolic or hypothyroid state. Granulomatous lesions considered are not only tuberculosis but also histoplasmosis, blastomycosis and actinomycosis. Other granulomatous occurring lesions are also discussed, as well as those complications seen in the larynx as manifestations of the collagen diseases. Maloney stresses the fact that being aware of systemic disease as a factor in laryngeal disease will put proper emphasis on the need for a careful medical history and general physical examination.

According to Thomas,[34] otolaryngologists rarely see tuberculosis of the oro-larynx, which is not secondary to pulmonary tuberculosis but is probably due to abdominal tuberculosis. Such a case is reported. Tuberculous reactions in the mouth and larynx are usually secondary to pulmonary infections and are considered due to implantation from expectorated sputum. The mode of spread in the case reported was probably hematogenous.

In a study of 131 cases of goitre Sonninen[35] found 22 cases of true tracheal compression without thyrotoxicosis and 23 cases of true toxicosis without compression. Significant laryngeal symptoms and signs of goitre were limited chiefly to the compression group. Principal symptoms were a con-

stant desire to clear the throat, lowering of the pitch of the speaking voice, impairment of quality of speaking voice and paresthetic sensations in the larynx.

Corbett[36] reminds us that the syndrome of Avellis consists of paralysis of the soft palate and larynx on the same side and partial paralysis of the constrictors of the pharynx. A lesion of the vagus nerve outside the jugular foramen can give rise to a syndrome such as described by Avellis. The symptoms presented are dysphagia, hoarseness or change in speech, while the clinical findings are paralysis of the soft palate with deviation of the uvula to the unaffected side. There is ipsilateral paralysis of the larynx with anesthesia of the palate and pyriform fossa. Such a case is reported.

Kyttä[37] comments on the sparse literature relative to acute epiglottitis. Seventeen patients with acute epiglottitis were seen from 1949 to 1959 at the Otolaryngological Clinic in Turku, Finland. Initially, all complained of severe sore throat and later increasing dyspnea. As a rule visualization of the vocal cords is extremely difficult because of the size of the epiglottis. Abscesses form frequently, and incision and drainage require good management to prevent aspiration asphyxia.

Hoarseness is a common complaint in tuberculous patients, according to Gupta,[38] and may be due to simple laryngitis or actual tuberculous involvement of a vocal cord. Hoarseness due to spontaneous recurrent laryngeal nerve palsy in tuberculous patients is not too common. Gupta reports 17 cases of palsy of the vocal cords in tuberculous patients and questions why most of these (14 cases) occurred on the left side. He quotes authorities on the course of the nerves and seems reluctant to accept the fact that the long course of the left recurrent nerve around the arch of the aorta and the apex of the lung, particularly a tuberculous lung, could well be the causative factor of paralysis of the left recurrent nerve.

Novik[39] reports the results of treatment of pseudo-croup with Prednisone(R) in every second patient seen. In the treated group no complications occurred, there was a favorable effect on the stridor, and the incidence of tracheotomy was reduced.

Leegaard[40] considers pseudo-croup a problem primarily for the laryngologist because of the importance of maintaining a free airway in the presence of acute laryngitis. The therapeutic measures most commonly used today are administration of relatively cool air with high relative humidity, corticosteroids, antibiotics and tracheotomy. Leegaard emphasizes the necessity of the pediatrician's watching the child's general care with special attention to fluid balance. This subject is covered in detail and must be read to be fully appreciated.

Forbes[41] states that disappearance of diphtheria respiratory viruses are predominant causes of obstruction of the airway. The characteristic manifestations of influenza are fever, headache, malaise, laryngo-tracheobronchitis and gastrointestinal symptoms, whereas in parainfluenza the onset is coryzal. In an adult parainfluenza is a minor cold, but in children it can be a cold, croup or pneumonia, and can even cause death. Because of secondary infection and the possibility of a bacterial infection, these patients should be treated with antibiotics.

According to Ers Estola and associates,[42] from 1951 to 1958, 794 patients with pseudo-croup were hospitalized in Helsinki. Of these, 18 died and 48 required tracheotomy in spite of antibiotics.

Cramblett[43] classifies croup into three broad divisions: infections, mechanical and allergic (angioneurotic) and then subdivides these. Factors predisposing to croup are age, sex, season, psychogenic, allergic and undefined. The viruses that cause croup are CA viruses, influenza virus (Hemadsorption I), adenoviruses, parainfluenza 3 hemadsorption virus type 2, CCA virus, and echo virus II.

In less than 20 per cent of cases, croup is due to Hemophilus influenza and Corynebacterium diphtheria; therefore, antibiotics seldom need be given. More emphasis should be put on general supportive measures, such as increased humidity, fluids intravenously, and sedation. If the condition worsens, tracheotomy should be done.

Bacteriologic investigation in croup is essential for proper management of a patient, even though only 14 per cent are due to bacteria, according to Ferris.[44] The viruses isolated were M. influenza and M. M. Parainfluenza types I, II, III. Ferris stated that 31 per cent of patients with croup had M. Parainfluenza virus.

Respirator treatment in cases of acute laryngotracheobronchitis is strongly advocated by Carlens, Widman and Norlander.[45] If dyspnea with inspiratory stridor persists after tracheotomy, there is risk of development of emphysema and pneumothorax. In patients with laryngotracheobronchitis, moistening of inhaled air is important, or else dried secretions and crusts form in the tracheobronchial tree and must be dissolved to some extent for easy removal. Dyspnea frequently develops, and inspiratory stridor entails increased respiratory work, which may bring on exhaustion. Carlens and associates emphasize the value of respirator treatment. They report two cases of infants whose lives were saved by judicious use of the respirator.

A progressive increase of obstructive laryngitis and laryngotracheal involvement was observed by Bauza and associates.[46] Laryngotracheobronchitis occurred more frequently and occasionally with a complication of measles. The development of dyspnea was an indication for immediate tracheotomy. This was performed on 149 children. The therapeutic value of corticosteroids in these children was questionable.

Agra and associates[47] report three cases of edema of the larynx. They briefly discuss the anatomy of the larynx and the pathology and causes of edema. All agree that when dyspnea develops, early tracheotomy is the treatment of choice.

INJURIES.

Dislocation of the crico-thyroid joint is a relatively rare condition. Knight[48] reports nine such cases. He describes the symptoms and therapy for successful reduction of these dislocations. Most of the dislocations were caused from

trauma, and automobile injuries formed the larger group. All laryngologists should read this excellent description of the individual cases, as textbooks contain little on this subject.

Early attention to laryngeal injuries is stressed by Cracovaner.[49] Particular attention should be directed to low tracheotomy and to reduction of any fractures or displacements of the laryngeal framework within the first few days. Open wounds should be immediately explored and repaired. Acrylic core molds or polyethylene tubes should be used when necessary to maintain an airway. The latter complications and their management are outlined in this concise but informative report.

Irvine[50] reports that while eating, a seaman was accidentally struck on the back by someone passing behind him. His head went forward and the handle of the fork struck the table forcing the fork into his mouth. A supralaryngeal swelling developed on the left side with some swelling of the arytenoids. He was hospitalized for several days, and it was concluded that the trauma to the larynx had been produced by the fork's digging into the posterior surface of the arytenoid region. No record of a similar case has been found in the literature.

Chadwick[51] believes that closed injuries to the larynx should be regarded as a separate entity from other types of laryngeal injuries, for treatment of these is distinctly different from the generally accepted principles of treatment in other types of laryngeal trauma. Eighteen cases of closed injuries of the larynx are reported. Laryngeal stenosis is uncommon, and as a rule, tracheotomy is not required. The literature is reviewed, with emphasis on the opinion of other laryngologists on this subject.

Automobile accidents continue to be the largest contributing cause of laryngeal trauma. Bennett[52] states that cartilaginous fractures of the larynx in general become increasingly difficult to reduce after 48 hours because of the rapid development of fibrous tissue and the difficulty in freeing the cartilaginous fragments. When the average patient reaches the otolaryn-

gologist for definitive treatment, about six weeks have elapsed, and there is no longer any possibility of manipulating the cartilage fragments. Bennett well describes treatment of fractures of the larynx but warns that each case presents a different problem so that no routine method can be used.

Problems of laryngeal injuries and stenosis are discussed by Suggit,[53] who reports five interesting cases of laryngeal trauma. He employed the generally accepted procedures to treat these injuries and obtained excellent results.

PARALYSIS.

Hawe and Lothian[54] state that the recurrent laryngeal nerve was exposed in 1,011 thyroidectomies with vocal paralysis in 28, of which three proved to be permanent.

In each case indirect laryngoscopy was done preoperatively, and direct laryngoscopy before the wound was closed at operation. If the nerve was paralyzed, the corresponding nerve would be examined very easily as it had been dissected free during the operation.

In two instances the nerve had been included in a ligature. In four to 12 months after the ligature had been removed, recovery took place. Many of the cases of temporary paralysis were due to trauma in exposing the nerve.

Paralysis that appeared in the early postoperative period was due to hemorrhage and fibrosis. These cases should be carefully watched, as they will probably prove to be temporary.

In several instances the tumor involved the nerve and, therefore, the nerve was sacrificed. Tumors that involve the nerve will produce paralysis, but by use of the exposure technique many nerves will be spared.

Priest and coworkers[55] performed arytenoidectomies, according to the Woodman technique, on three children because of bilateral abductor paralysis. Two were suffering from bulbar poliomyelitis, and the other had paralysis of the bilateral recurrent nerves of unknown origin. In all cases the arytenoidectomy permitted elimination of the tracheotomy

tube and closure of the tracheostomy. Priest and associates found few references in the literature to arytenoidectomies in children. Your reviewers shall soon report a case of bilateral abductor paralysis resulting from a foreign body lodged in the postcricoid area for at least three months and causing bilateral abductor paralysis. Seven years later an intralaryngeal arytenoidectomy was performed, and the child was successfully decannulated.

In a recent study of 115 cases of recurrent laryngeal nerve paralysis of known cause, Williams found that 22 were bilateral; 16 followed thyroidectomies, two were due to peripheral neuritis and four to carcinoma of the esophagus and thyroid gland. Robertson[56] reports a case of acute bilateral laryngeal nerve paralysis in an elderly patient. Autopsy disclosed abscess of the esophagus with acutely inflamed nodes in close relation to both the right and left recurrent laryngeal nerves. Pressure from these nodes on these nerves undoubtedly was responsible for the paralysis.

Paralysis of the left recurrent laryngeal nerve secondary to cardiac enlargement is an infrequent complication. Such a case is reported by Corbett.[57] The paralysis was thought to be due to an enlarged left ventricle pressing on the left recurrent laryngeal nerve. In many cases of advanced mitral stenosis the pulmonary artery is atheromatous. Corbett reminds us that it is accepted by most observers that an atheromatous aorta is the most common cause of left recurrent nerve paralysis. This being so, it is a logical conclusion that the pressure of an atheromatous vessel, combined with the pull and drag of a hypertrophied heart, can cause damage to the nerve producing paralysis.

Smith[58] reminds us that recurrent laryngeal nerve paralysis, presumed to be due to malignant infiltration, is an absolute contraindication to operation in carcinoma of the bronchus. When a decision of such supreme significance hangs on a single physical sign, it is of the greatest importance that the most meticulous care and accuracy be employed in the performance of mirror laryngoscopy, for upon this critical observation the patient's life depends.

OBSTRUCTION.

Leonard[59] discusses the mechanics of laryngeal collapse secondary to partial upper respiratory obstruction in certain selectively bred dogs. The partial obstruction in brachycephalic dogs due to the exaggerated length of the soft palate interferes with inspiration and increases the negative pressure in the larynx. After an extended period the ventricles evert into the lumen of the larynx, followed by the cuneiform tubercles and finally the corniculate tubercles. Surgical excision of these obstructing masses relieves the obstruction. All laryngologists should find these descriptions stimulating and possibly of practical value in treatment of similar conditions in the human larynx.

Abbey[60] advocates laryngostomy for immediate relief of sudden laryngeal obstruction in adults. He defines laryngostomy as an incision through the skin and cricothyroid membrane, opening into the subglottic region. The procedure is simple and safe, can be easily and precisely performed, and is virtually free from complications. A transverse incision is used, and the cricothyroid membrane is incised. Abbey mentions that even in skilled hands the extremely urgent tracheostomy with its complications can be exceptionally difficult.

Laryngostomy is to be used for temporary relief of obstruction only and must be immediately followed by a well organized tracheotomy. Laryngostomy is not suitable for children, because their larynges are small and their vocal cords lie near the site of the operation.

Production of an emergency airway is simple in principle, and relatively so in practice, although it can be one of the most difficult surgical procedures. With this in mind, Ruhe, Williams and Proud[61] made an exhaustive study of emergency tracheotomies. Because there is no generally accepted "best" method of entry into the airway, they reviewed the various methods recommended. They discuss the virtue of these methods and present illustrations of all instruments devised for rapid entrance into the airway. The generally

accepted method for a dire emergency is performance of cricothyrotomy, and later routine tracheotomy can be performed in a hospital under aseptic conditions.

STENOSIS.

Management of an intractable stricture of the upper trachea in a child aged 14 months is described by Borrie.[62] This stricture developed after a high tracheostomy for edema of the glottis after the removal of an intralaryngeal foreign body and an episode of bronchial obstruction requiring 36 bronchoscopies. Treatment consisted of lowering the tracheostomy, skin grafting the strictured area at the level of the first tracheostomy, and the wearing of a specially designed polyethylene tube for six months.

Lynch and LeJeune[63] offer a new method to correct web formation between the vocal cords. Under suspension laryngoscopy a small opening is created in the web at the anterior commissure, and a polyethylene tube is inserted into this opening. This tube is held in place by a silver wire inserted through the thyrohyoid membrane, through the tube, and emerging through the cricothyroid membrane. The wires are held externally by appropriate buttons. An epithelized opening is created at the anterior commissure, and when completely healed the web is excised along the edge of the cord.

We are in total agreement with Bennett[64] that treatment of laryngeal stenosis is usually difficult. In the past, diphtheria and high tracheotomies were largely responsible for strictures; today, trauma and automobile accidents are the most frequent causes. Bennett uses the Erich method for skin grafting, and the rubber mold is removed on the tenth day. An acrylic obturator is then inserted with the larynx and attached to the tracheotomy tube and remains in place as long as six months. Decannulation can eventually be obtained in almost all cases of laryngeal stricture if the surgeon is persistent and patient.

Imperfect techniques of tracheotomy are believed by Fox[65] to be responsible for most cases of laryngeal stenosis. Too

high a tracheal incision, removal of elipse of tracheal ring, and unnecessary dissection, are chief defects of techniques. In all of Fox's cases of chronic stenosis the cicatricial tissue was excised through an external laryngostomy. Because of their low tissue reactivity acrylic stents were felt to be the most successful form of internal support and were allowed to remain in place with silver wire suture for four to 16 weeks. Success in establishing an airway was achieved in 14 of 15 cases. This technique appears to offer a fairly reliable solution to the difficult problem of laryngeal stenosis.

Norris's[66] discussion of acrylic molds in chronic laryngeal stenosis is excellent. He considers acrylic material ideal for the larynx, because it is non-irritating, non-porous, easily shaped and tolerated with comfort even for months. Increasing edema of the arytenoid eminences, dysphagia, tenderness, swelling or external redness usually indicates a response to a mold that is too large. The usual duration for use of the acrylic mold is four to six months, but this depends largely on the degree of stenosis present.

Rasmussen[67] described management of a complete laryngeal stricture resulting from lupus. During convalescence from lupus a complete stricture developed in the larynx of a 39-year-old woman. Ten years later the cords were found to be completely adherent. Preoperative and postoperative treatment with dihydrostreptomycin was instituted, and through a laryngofissure approach, the interior of the larynx was exposed. The fibrous tissue responsible for the stricture was extirpated, and a nylon sponge covered by skin graft was inserted and left within the larynx for four weeks. Good results were obtained with mobility of left cord.

Congenital stenosis of the cricoid cartilage is a rare condition; however, France and Stirling[68] report two such cases seen within ten days. Both patients had esophageal atresia and tracheo-esophageal fistulas, and neither survived operation. A good discussion is presented, and a warning is sounded that the possible occurrence of cricoid stenosis tends to increase the risks associated with intubation of the newborn.

ATRESIA.

Martin and Hogg[69] call attention to the fact that within the past 20 years, the outlook for infants with esophageal atresia and tracheo-esophageal fistula has changed from one of complete hopelessness to hopefulness with anticipated cure of approximately 85 per cent. Serious associated anomalies may be expected in approximately one-third of the cases, and their early recognition and treatment are obviously of paramount importance. Martin and Hogg report a case of esophageal atresia associated with chondromalacia and describe their method of handling the case successfully. Occurrence of respiratory distress out of proportion to the degree of pneumonia present should arouse suspicion of an associated anomaly such as chondromalacia.

SPASM.

Severe laryngeal spasm still occurs occasionally, and may at times be an alarming emergency. Atropine as a prophylactic is of some value. Rosen[70] cites Burnstein, Lee, Goodman and Gilman as advocating atropine for laryngeal spasm. Based on the results of experiments on use of atropine in laryngeal spasm, Rosen is forced to conclude that this drug in clinical doses is of no therapeutic value in this condition.

STRIDOR.

Goldbloom and Dunbar[71] report a case of a four and one-half-month-old boy with congenital stridor due to premature calcification of cartilage in the larynx and trachea. In reviewing the literature, they found only three cases. In their case a possible genetic etiology was entertained, as the mother showed laryngeal and tracheal calcification.

BENIGN TUMORS.

Asymptomatic solitary masses in the neck pose a diagnostic challenge to the physician. Tamoney[72] saw a patient with a stony hard, non-tender mass measuring 3 by 2 cm., in the neck. It moved with swallowing and appeared to be replacing the upper pole of the right lobe of the thyroid gland. The

preoperative diagnosis was solitary nodule of the right thyroid gland strongly suspicious of carcinoma. At operation the tumor proved to be a chondroma attached to the right external wall of the cricoid cartilage. Tamoney comments on the rarity of these tumors and presents a brief review of the literature on this subject.

A case of fibrous dysplasia of the trachea and larynx is reported by Engelking,[73] who was unable to find any cases of this benign lesion in a review of the literature. This dysplasia, which was present in the subglottic portion of the larynx and the upper portion of the trachea, produced considerable obstruction of the airway. Removal followed by skin grafting was successful.

In reviewing the literature, Yurich and Beekhuis[74] found eight cases of multiple neurofibromatosis with associated involvement of the larynx. They report one case. The family history in all of these cases points to a hereditary tendency of this disease. Surgical excision from the larynx appears to give satisfactory results.

Amyloid tumors of the larynx are rare lesions which are frequently associated with rheumatoid arthritis. Such a case is reported by Leitch.[75] Surgical removal of the tumor afforded complete relief. The presence of amyloid is thought to be due to abnormality of protein metabolism.

Gerwel and Nawrocki[76] state that true angiomas of the larynx are relatively rare, and lymphangiomas of the larynx are extremely rare. Only a few cases have been found in a review of the world literature. Lymphangiomas of the larynx appear chiefly in regions where friction occurs and where there are numerous lymph vessels. Seldom are the true cords involved, and when they occur on the aryepiglottic folds, they attain considerable size and are more or less pedunculated. The histology of this tumor is discussed in detail, and a case of lymphangioma operated on successfully is reported.

Angiomas are common tumors of childhood but occur only rarely in the larynx. Cameron and coauthors[77] report three cases. These patients presented similar symptoms of laryn-

geal obstruction, and in spite of early tracheotomy and conventional medical treatment, all died. The difficulties in diagnosis, and the relative merits of surgical and radiotherapeutic treatment are discussed.

El Mofty[78] described an interesting case of removal of a large, benign, supraglottic cyst having both intralaryngeal and extralaryngeal extensions. An external incision was employed, and by dissection through the thyrohyoid membrane, the internal portion was removed with difficulty. The cyst was thought to be of congenital origin.

Stewart[79] found less than 20 cases of adenoma of the larynx in the literature during the past 30 years. The etiology of these tumors is obscure. A case report is presented in which a cystadenoma was found attached to the posterior surface of the larynx at the level of the cricoid cartilage. Removal by laryngofissure was accomplished without difficulty.

Walter[80] adds two cases of granular cell myoblastoma of the larynx to the 19 already reported. Granular cell myoblastomas, although still uncommon, should not be classified with the rarest tumors. The exact histogenesis has not been definitely determined, but several theories (myogenic, histocytic and neurogenic) have been proposed to explain their origin. Walter states that myoblastomas, as they occur in the larynx, are usually small, firm and nodular. The usual symptom produced is hoarseness, and local excision suffices. Walter agrees with other reports that in the larynx these tumors are always benign.

Beekhuis[81] was able to find reports of 16 cases of granular cell myoblastoma of the larynx, to which he adds three more. As can well be surmised, the tumor is rarely found in the larynx. Grossly, the lesions are small nodules or raised plaques, usually not more than a few centimeters in diameter, with a pale appearance. Beekhuis states that although little is known of the etiology, it is thought that degeneration of the striated muscle contributes to their development. Most myoblastomas have been found on the posterior portion of

the vocal cords. Myoblastoma of the larynx is a benign lesion, but Beekhuis stresses the importance of differentiating it from carcinoma.

Because of the rarity of myoblastoma of the larynx Balshi[82] made a study of these unusual tumors encountered at Jefferson Medical College Hospital during the past ten years. Hoarseness is the usual symptom. The various etiologic theories are discussed. Balshi states that most reported granular cell myoblastomas are histologically and clinically benign. Treatment is local excision. Five cases seen during the ten-year period are reported in detail.

Rock and Fisher[83] state that squamous-cell papillomas are common epithelial neoplasms of the mouth and larynx. Those of the larynx that occur in children are inherently benign and multiple, and tend to regress at puberty. It has been suggested that these lesions may be viral or related to hormonal influences. In the adult they are considered by some as premalignant neoplasms. Rock and Fisher report three cases of an unusual form of squamous-cell papilloma which they designated as florid papillomatosis of the larynx. Recurrences are frequent. The macroscopic and histologic appearance of the neoplasm may erroneously suggest malignancy; however, absolute morphologic criteria for the diagnosis is lacking, and metastasis has not been observed, although two cases have been followed for six and 11 years.

Butler and Goff[84] define a laryngocele as a cystic dilatation of the saccule or appendix of the ventricle of Morgagni with classification determined by extent of development into internal, external or combined types. Symptoms are discussed. A case is reported of an internal laryngocele successfully excised endoscopically followed by fulguration; 11 months later radiographic examination showed the larynx had returned to normal.

The value of roentgenography is stressed by Lund,[85] who employed this examination to confirm his diagnosis of an internal and an external laryngocele. Surgical removal was

successfully accomplished, but four months later a suspicious growth in the larynx, diagnosed by biopsy as carcinoma, necessitated laryngectomy.

A 45-year-old man had a swelling in the side of his neck accompanied by discomfort in the throat and vocal changes. Cataldo[56] found the mass resistant to pressure and noticed it increased in size on forced inspiration. Direct laryngoscopy failed to establish a diagnosis. Roentgenography showed an area of infiltration and an enormous air-pocket with classical characteristics of a laryngocele. This case is of interest because it demonstrates the value of radiography in establishing the presence of a double lesion.

Galloway, Soper and Elsen[57] sound a note of warning in the use of irradiation in benign lesions. Immediate results are often spectacularly good; however, late serious complications may result from irradiation. It has been established that irradiation may cause leukemia, carcinoma of the skin, carcinoma of the thyroid and other organs. Galloway and associates state that at least six cases of intralaryngeal carcinoma have been reported as a result of irradiation in multiple papillomatosis of the larynx. A case is reported of an eight-year-old boy in whom multiple papillomatosis of the larynx developed. After repeated removals, radium was used intralaryngeally. Subsequently carcinoma of the larynx developed. This is a most excellent article, sounding a note of warning in the use of irradiation in benign cases, and should be read to be appreciated.

MALIGNANT TUMORS.

Kim[58] presents a statistical study of 200 cases of benign and malignant tumors of the larynx, taken from the records of laryngoscopic clinics of five large hospitals in Korea, covering a period of five years. The distribution according to age and sex was found to be closely similar to that in this country.

Nobre[59] presents the results of the study for clinical staging of cancer of the larynx, based on the evaluation of various propositions and recommendations sent to the "Committee on

Clinical Stage Classification and Applied Statistics" by consulted specialists and by international organizations also engaged in solving this problem. The suggestions as presented by Nobre give promise of much success!

Determination of the nature and site of a neoplastic lesion by means of radioisotopic techniques is generally accomplished by using radioactive substances. In experiments Filippi, Ferrini and Cremonesi[90] used radiophosphorus (P^{32}) because neoplastic tissue takes it up in greater percentage than do normal cells, owing to the more active phosphorization processes in neoplastic cells. Patients having malignant lesions of the larynx and pharynx were given P^{32} orally or intravenously. Radioactivity counts in neoplastic homogenized tissue were higher than those in control normal tissue. It was concluded that P^{32} studies provide additional information helpful in making a preoperative diagnosis of malignant tumors of the larynx and pharynx.

Routine use of cinefluorography in every laryngeal or hypopharyngeal malignant tumor, in order to help determine the size, extent and degree of invasion of the tumor, particularly in areas difficult to visualize endoscopically, is described by Kirchner, Scatliff and Shedd.[91] An even more interesting application of cinefluorography is in postoperative investigation of complications. The same modality offers a satisfactory means of studying voice production in patients who have been operated upon by various modifications of laryngectomy or laryngopharyngectomy.

In an excellent article Maloney[92] describes the pitfalls in the diagnosis of cancer of the larynx. He states that early diagnosis in laryngeal cancer must start with an appreciation of the importance of minimal complaints referable to this organ. He stresses the important fact that there are hidden areas of the larynx that increase the difficulty of a thorough examination. Complications, such as seeing a laryngeal lesion for the first time after biopsy by a colleague, or examination of a larynx after roentgenotherapy, invariably accentuate diagnostic difficulties. He correctly pleads for close liaison

between pathologist and laryngologist, and we heartily concur that many patients are better examined by direct laryngoscopy after induction of general anesthesia.

Maher[93] presents an interesting, extensive study of surgical material from 73 cases of papilloma of the larynx and 340 cases of carcinoma of the larynx, seen in the Department of Pathology at the University of Michigan during a ten-year period. All but four of the 73 cases of papilloma were of the juvenile type and the majority required multiple operations. Three of the papillomas showed progression from typical juvenile papillomas to carcinomas. The fact that carcinomas may arise in papillomas forces Maher to advocate continued clinical surveillance and histologic examination of such lesions; this is particularly true for papillomas in adults.

The 340 cases of carcinoma of the larynx were graded, the majority being placed in Grade II and III. Only seven were reported as carcinoma *in situ*. The poorly differentiated carcinomas of the larynx occurred in a slightly older age group than did the well differentiated carcinomas. Maher also included in this study a review of 14,000 necropsies performed from 1923 to 1956; 47 carcinomas of the larynx were encountered in this group.

The five-year survival rate of intrinsic cancers of the larynx, when correctly treated, is 70 to 80 per cent, whereas it is only 25 to 40 per cent for extrinsic cancers of the larynx, epiglottis, false cords, ventricles and subglottic regions according to McCall, Hendershot and Whitaker.[94] This article contains an excellent summary of the fine points in diagnosis and therapy of the various types of carcinoma with particular emphasis on the location of the lesion. Definitive treatment must be outlined and adhered to, for McCall and associates firmly believe that it is a fallacy to suppose that if one form of treatment does not succeed, other measures, such as laryngofissure or laryngectomy with or without neck dissection, may then be instituted. The laryngologist who first decides the mode of treatment for the patient with cancer of the larynx determines whether the patient will live or die.

Tapia Acuna[95] expresses his views on the various types of carcinomas of the larynx and his method of management. He calls particular attention to the possibility of thyroid gland involvement indicated by enlargement of the thyroid cartilage in the presence of carcinoma and reports two such cases. A paramedian skin incision prolonged to the opposite side above and below is advocated. This exposure also serves for neck dissection.

The "cylindroma" is a slow-growing adenocarcinoma of the salivary gland type that occurs occasionally in the trachea and larger bronchi and less commonly in the larynx. Suehs[96] states that this tumor has been given various names including mixed tumor of the salivary gland type, basal cell carcinoma, and adenocystic carcinoma; but the term, cylindroma, is the oldest and most descriptive of the histologic pattern. Suehs analyzed 32 cases of tracheal and six cases of laryngeal cylindroma in the literature and added two cases of his own. His case of cylindroma arising on the vocal cord is the first such case to be reported. The results of treatment of cylindromas are not satisfactory. Five-year results are meaningless in view of the high incidence of delayed local recurrence and distant metastasis.

Catlin[97] briefly discusses carotid artery ligation, beginning with Pare's first recorded operation 400 years ago. He reports the case of a patient with cancer of the extrinsic larynx, who, during two years of surgical treatment, survived bilateral common carotid artery ligation, the first in 1956 and the second in 1958. It is apparent that the vertebral arteries alone supply adequate circulation to the brain. Catlin emphasizes the importance of avoiding cerebral hypotension at the moment of carotid ligation.

Stout[98] correctly states that there are stages of cancer of the larynx in which there is no obvious mass or invasion of cartilage or deeper soft tissues. Such is carcinoma *in situ* of the larynx, in which occasionally the vocal cords show little or no change. The cord may appear slightly red or rough, and on biopsy, carcinoma *in situ* will be found in one small

patch or in multiple areas. Stout reports 48 such cases, in 29 of which there was no invasion. He considers radiotherapy the preferable form of treatment.

Early cancer of the larynx is discussed by Frazell and Gerold,[99] who state that cancer may arise at any point on the mucous membrane of the intrinsic larynx, the extrinsic larynx, or the ill-defined region referred to as the laryngopharynx. Only under exceptional circumstances are these latter lesions diagnosed early. Early cancer of the larynx is described as a lesion confined to one vocal cord only, or one with limited involvement of the anterior commissure and opposite cord.

Partial laryngectomy was performed on 194 patients with successful results in 74.7 per cent. What is meant by partial laryngectomy is left to the discretion of the reader.

Treatment of malignant disease of the larynx and laryngopharynx is a controversial matter of world-wide interest. The merits of surgical excision and of radiotherapy have been hotly debated; but in Britain a measure of agreement, according to Korkis,[100] is gradually emerging. Much importance is placed on the anatomic site and clinical staging of carcinoma of the larynx. According to Korkis, radiotherapy is used extensively in the treatment of cancer of the larynx, but for recurrences, surgical treatment is employed. From a study of the material presented it would seem that cancers of the larynx treated by early excision fared better than those treated by radiation first. This is an extensive paper that must be read to be appreciated.

Rigual, Witter and Barnhill[101] reviewed 129 cases of laryngeal carcinoma encountered at the Oklahoma University Hospital and the Wesley Hospital from 1941 to 1958. An attempt was made to analyze some of the factors that might influence the prognosis. Survival time was correlated with presence or absence of metastasis, location of the primary lesion and various methods of therapy. Only 3.9 per cent of the series were Negroes. It is regrettable that all follow-up statistics were recorded as being "alive without recurrence at least

one year after therapy." More determined efforts should have been made to obtain accurate five-year follow-up in order to evaluate the therapeutic procedures more accurately.

Hoffmeister and Glidewell[102] present an interesting study of radiation and surgical treatment of carcinoma of the larynx. The results of surgical treatment were much better than those of irradiation. For example, in intrinsic cases, the recurrence rate was 16 per cent for surgical cases and 47 per cent for irradiated cases. In extrinsic cases these rates were 30 per cent and 71 per cent, respectively. These authors conclude that the improvement in the five-year survival rates of patients treated by radiation was statistically insignificant and clinically negligible.

A small localized squamous cell carcinoma on the edge of a vocal cord is more amenable to cure than any other cancerous lesion except basal cell carcinoma of the skin. McCabe and Magielski[103] point out that the reason for this is largely that the lesion produces early symptoms, is easily visualized, and is surrounded by a paucity of lymphatics. The five-year cancer cure by total laryngectomy has remained static during the past 20 years regardless of surgical innovations, such as wide-field over narrow-field laryngectomy. In small glottic carcinomas of the vocal cords, McCabe and Magielski believe that irradiation and surgical excision offer about the same results. Their five-year cure rate was 83 per cent by excision and 78 per cent by radiation. If the services of a highly competent radiologist are available, the final results will be good. They warn, however, that radiation is fraught with certain risks, and a good voice after such therapy cannot always be guaranteed. The number of failures after total laryngectomy will decrease only with proportionate increase in patient and physician education.

We are told by Shaheen[104] that cases of malignant melanoma of the larynx, both primary and metastatic, are rare, only four cases having been reported. Shaheen adds a fifth case. The patient originally complained of a painless swelling on the left side of the neck for three months and persistent hoarseness for seven weeks. The lesion, located on the ante-

rior surface of the left arytenoid, was removed at the same time as block dissection of the neck was performed. The patient remained well for three years.

In order to obtain a uniform radiation dose over a growth within the larynx, Kaae and Brene[105] adopted use of wedge filters, because there is less irradiation to the surrounding healthy tissue. Special attention must be given to use of small fields in connection with this technique in the treatment of small carcinomas of the vocal cords.

Goldman and Silverstone[106] state that 35 years' experience with radiation therapy for carcinoma of the larynx has demonstrated its efficacy in their hands. They report astounding results in a group of cases of carcinoma of the larynx. The five-year survival rate, in patients with carcinoma limited to the vocal cords, was 88 per cent; in those with lesions of the intrinsic larynx suitable for laryngectomy, it was 63 per cent. It was 56 per cent in patients with carcinoma of the anterior group of the intrinsic larynx, 32 per cent in those with carcinoma of the posterior-lateral group of the intrinsic larynx, and 14 per cent in patients with the extensive group of the intrinsic larynx. They advocated combined radiation and surgical therapy for lesions with a poor prognosis.

In a discussion of radiation therapy of carcinoma of the larynx, Carpender[107] states that the choice of treatment between operation and radiation must be based on many factors. He frankly admits that in subglottic lesions radiation has little to offer, whereas radical excision gives the patient a fair chance. Carpender describes the technique used in radiation therapy. Among 68 patients whose primary treatment was radiation, there were "26 known to be alive and free of disease, an absolute cure of 38 per cent." It is not stated whether this is a five-year cure or less. Surgeons would consider 38 per cent cures in selected cases poor results. It will be interesting in a few years to have a follow-up report by Carpender, as he states that the University of Chicago Clinics follow closely the desirable pattern of collaboration between radiotherapist and otolaryngologist. Reports of their final results will be valuable.

Radiation therapy has made considerable progress in the last two decades, according to Chu.[108] Much of this progress has been due to an increased knowledge of fundamentals of radiation physics, radiobiology and improvement of facilities and equipment. Chu correctly states that, as a rule, radiation therapy offers better functional results in carcinoma of the larynx when cures are obtained. Her statement, however, that results obtained with laryngofissure and radiation therapy are equally good is challenged by laryngologists. Chu further states that in advanced cases "laryngectomy may give better results, but functional disabilities follow." This latter is far preferable to a ceasing of all organic functions.

Black[109] reports the results of local radium treatment in 77 patients with carcinoma of the larynx. The Finzi-Harmer method was employed. An external incision exposed the outer surface of the thyroid ala. Adequate cartilage was removed, and the standard 1 mg. content radium needles were inserted under the edges of the window made in the thyroid ala and left in from three days to one week. The cure rate was 72 per cent, but because of complications, such as radionecrosis, and the notable advances of tele-radiation, this method of local radiation treatment has been abandoned.

Rounthwaite and Greenway[110] report a case of carcinoma of the left pyriform sinus with extension into the larynx. Cobalt therapy was administered, and six months later, laryngectomy with block dissection of the neck was performed. They also made a survey of the first five-year results of Cobalt therapy at Victoria Hospital. As a result of this survey, they state that from now on they will treat more patients with cancer of the larynx surgically.

Jesberg[111] reviews the history of the development of laryngectomy from 1870 to the present day. All engaged in this particular field of work contributed their bit, and the sum of their contributions represents the current surgical treatment of diseases of the larynx. With the advent of the antibiotics, postoperative fistulas and other complications decreased. Jesberg advocates wide field laryngectomy with removal of the strap muscles, hyoid bone, larynx, pre-epi-

glottic space and upper tracheal rings in one specimen. Tribute is paid to Schall for his many contributions to laryngeal surgery. In predicting the future of laryngectomy, Jesberg says that our vain hope is to resolve the nature of cancer at a fundamental and etiologic level, but this is to some extent hazardous. The continued and expanded use of primary laryngectomy combined with neck dissection, both prophylactic and curative, should improve survival rates and may add 10 to 20 per cent to the 60 to 65 per cent five-year survival rate of wide field simple laryngectomy.

Nundy[112] describes his experiences in the treatment of cancer of the larynx in Burma. He comments on the woeful ignorance of the population in general, relative to symptoms of cancer. Most patients with cancer of the larynx in Burma are seen when the disease is far advanced, as borne out by the fact that of 104 patients seen, only 33 were treated by laryngectomy between April 1954 and March 1957. Because of existing conditions postoperative complications were numerous and serious; however, Nundy considers the results obtained very gratifying under existing conditions.

Butler and associates[113] describe a new type of cutaneous incision for laryngectomy, which was observed by one of them in a modified form, in London, where it was used with much success by Prof. F. C. Ormerod. An incision is made on both sides of the neck, beginning at a level of the hyoid bone, and is carried longitudinally downward and obliquely along the anterior border of the sternocleidomastoid muscle to about the level of the cricoid cartilage. The convex extension of this incision at the distal end is made to outline a circle of skin whose radius approximates a tracheal stoma and is maintained in continuity with the outlined apron flap. The advantages described and the enthusiasm of these authors for this new procedure have created a desire in the reviewers to use such an incision on the next laryngectomy to be performed by them.

Partial laryngo-pharyngectomy in continuity with radical neck dissection with preservation of laryngeal and pharyngeal function in selected lesions of the larynx and hypopharynx is

advocated as the treatment of choice by Ogura, Jurema and Watson.[114] In an excellent paper they report successful results in selected lesions of the aryepiglottic fold, medial, lateral and anterior wall of the pyriform sinus and lateral wall of the hypopharynx. Preservation of the voice combined with adequate excision of the laryngeal or laryngopharyngeal malignant lesion has been the goal of surgeons for many years. To employ this conservative procedure, exact preoperative localization of the tumor is necessary. Surgical technique is adequately described, and because the incidence of cervical metastases for pyriform sinus cancer is estimated to be from 40 to 60 per cent, radical neck dissection is mandatory. All interested in this type of surgery should read this excellent article.

The widespread use of radical neck dissection in the treatment of carcinoma of the larynx is well established, according to Reed and Snow,[115] but they state that the indications for this procedure are uncertain. They were impressed with the relatively poor survival rate in practically all patients in whom a metastatic node was found. They undertook a study of 75 cases of radical neck dissection to show the importance of the site of the primary lesion, the number of nodes and the size of the nodes. They concluded that in patients having nodes 2 cm. or smaller the prognosis was good, whereas if the node was 4 cm. or more, the prognosis was poor. Patients with small single nodes have the best chance of survival if radical neck dissection is performed as early as possible.

Evaluation of laryngectomy with radical neck dissection forms the basis of an excellent presentation by O'Keefe.[116] Carcinoma of the larynx of extra-cordal origin, or of cordal origin but with gross extension to adjacent areas, is more malignant than cancer confined to the true vocal cords. In 28.9 per cent of cordal lesions, thought ideal for laryngectomy, malignant metastasis to regional lymph nodes subsequently developed. O'Keefe advocates prophylactic neck dissection in all lesions extending beyond the vocal cords even though cervical lymph nodes are not palpable. Among 68 such cases (23.5 per cent) positive nodes were found in 16.

When cervical nodes are palpable in conjunction with a lesion of the larynx, radical neck dissection is mandatory.

Montreuil[117] presents an interesting article covering all phases of cancer of the larynx. He follows the generally accepted rules for the management of the various types of laryngeal cancer and offers a new thought in care during the immediate postoperative period. He dislikes use of Levine nasal feeding tubes, which, he thinks, might occasionally be the cause of irritation and infection of the hypopharyngeal mucosa. Instead, he has devised a new method whereby a feeding tube is brought out through a button-hole incision made in the upper part of the esophagus through the lateral part of the neck. This method has proved most satisfactory, and the patients are more comfortable without the nasal tube. The tube is usually removed on the sixth postoperative day. No fistulas have resulted from this procedure. This paper presents a new thought, and only time will prove its worth.

The return to the natural mode of nutrition as quickly as possible after laryngectomy has been an intriguing problem to Miodoński.[118] After 30 years of experience with this type of work, he has adopted a procedure that has given more successful results than any previously used. He has arrived at the conclusion that any type of feeding tube, large or small, is a source of great danger to the pharyngeal sutures. To avoid this, no feeding tube is used initially. For the first three days the patient is nourished parenterally. From the third day on a feeding tube is introduced through the nose and remains in place only when nourishment is given. After the seventh day the patient is allowed to eat normally.

Pulido[119] describes the case of a patient in whom tachycardia developed on the seventh day after laryngectomy. The heart beat was 150 per minute. This complication was attributed to development of a hematoma in the vicinity of the pneumogastric nerve.

Simpson[120] describes a cleverly conceived one-stage pharyngo-esophago-laryngectomy for post-cricoid malignant tumors. It consists of removing the pharyngo-esophageal cuff

with the back half of the larynx and trachea. The upper portion of the severed trachea is attached to the skin, and the anterior half of the larynx is moved backwards against the posterior pharyngeal wall. The lower portion of the severed trachea is anastomosed to the upper portion of the esophagus, forcing what remains of the larynx to play the role of the upper portion of the esophagus. Simpson states the operation is not difficult, and the results are gratifying.

Pap[121] stresses the importance of selecting for each individual case, a definite preoperative plan for reconstruction of the pharynx. He advocates primary reconstruction, using a skin graft supported by a Portex self-retaining tube whenever possible. If local tissue factors are not favorable, however, multi-staged procedures utilizing either sliding flaps from the neck, or acromio-thoracic tubed pedicles are indicated. Pap believes that formation of strictures can be prevented by use of the Moore-Faulkner method of introducing a portion of the skin flap into the pharyngostoma and into the esophagostoma. A case is reported which illustrates nicely the difficulties of such work and offers helpful suggestions to any surgeon doing similar types of cases.

The attitude of an optimist must be assumed, says Harris,[122] when dealing with large advanced carcinomatous lesions of the laryngopharynx and adjacent cervical esophagus, for at best, the five-year survival rate is poor with either surgical treatment or irradiation. Eight cases of skin grafting to the pharynx and cervical esophagus for extensive malignant lesions are reviewed. The technique used is described. Harris prefers the Edwards Tapp tube as a stint. He warns that skin grafting in order to preserve an ample tube for swallowing in cases of partial or complete laryngopharyngectomy and cervical esophagectomy is not without its complications and hazards.

It is estimated that 5,000 people get cancer of the larynx annually and that approximately 50 per cent of these undergo laryngectomy. Gilmore[123] pleads for early rehabilitation of these patients, because many of them are still wage earners. The need for social and vocational adequate speech is great.

Gilmore discusses the personal problems that frequently compound the patients' difficulties, speech training and social adjustments.

Silverberg[124] writes about the plight of laryngectomees whose most conspicuous concern naturally is the inability to speak. He dwells on their trials and tribulations and strongly advocates association with groups such as the "Lost Chord" Clubs. In this atmosphere these patients can share and compare their experiences, ideas, attitudes, fears and anxieties with each other.

Svane-Knudsen[125] states that modern authors generally agree that the esophageal voice is formed by a pseudoglottis consisting of muscular-mucous folds or narrowings in the hypopharynx, set into vibrations by air streaming from a reservoir in the esophagus; through the pseudoglottis; and further, through the normal resonance cavities of the mouth and nose. This human language development in this curious manner is still the subject of some divergence of opinion. Knudson noted that elderly patients have more difficulty developing esophageal speech; of 60 patients 18 were unable to develop an esophageal voice.

A survey by Johnson[126] of all laryngectomized patients in Veterans Administration Hospitals in the United States brought forth many interesting facts. Particular attention was directed toward the history, symptoms and speech development of all laryngectomees.

The importance of preoperative talks and reassurance of the laryngectomee that he can attain esophageal speech is stressed by Fontaine and Mitchell.[127] Friendly encouragement by good esophageal speakers at this period is useful. Postoperatively, as soon as the nasogastric tube is removed, production of esophageal sounds is encouraged. Group and individual instruction is utilized in efforts to deal with the emotional factors in the acquisition of an esophageal voice. Fontaine and Mitchell believed that postoperative adjustment of each patient is related largely to his previous adjustment. Several detailed cases are described in this informative presentation.

Personal experience has no equal. Perrin Long[128] discusses from first hand knowledge the problems, physical and psychic, that confront a laryngectomee. In an editorial he describes the many problems and frustrations faced by a laryngectomee. Although esophageal speech is highly desirable, all laryngectomees cannot master this method of communication.

The Bell Telephone Laboratory recently perfected a new type of electrolarynx, which Long highly recommends. It is reasonably priced so that laryngectomees desiring such an instrument may easily obtain one. This excellent editorial by an eminent physician who lost his larynx should be read by every laryngologist.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct only one examination in 1962, and this will be held at the Palmer House in Chicago, Illinois, October 28-November 1st.

TYMPANOSCLEROSIS—A REVIVED CLINICOPATHOLOGIC ENTITY.*

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INTRODUCTION.

The present era of microsurgery of the temporal bone has led to a revival of interest in the pathological anatomy of the middle ear. There has been a tremendous increase in aural surgery in recent years, due largely to the newly developed reparative surgical procedures. The successful surgical repair in otosclerosis and tympanomastoid disease has stimulated the development of improved instruments and techniques. The use of antibiotics to combat infection and to insure proper healing has increased the percentage of good results. In the course of stapes mobilization surgery, and even more in the operations for repair of the sound conduction mechanism, there is being observed microscopic pathology that has been ignored or overlooked during the past half century. There has been an astonishing amnesia amongst otologists who "forgot" stapes mobilization for half a century and it is now evident that they "forgot" tympanosclerosis for even longer. The vast majority of experienced otologic surgeons who have been doing many radical mastoidectomies say they never saw a case. One must assume that this condition has been repeatedly mislabeled and probably called cholesteatoma.

In the course of this paper, 16 cases of tympanosclerosis operated upon by the author and his two associates in the 34 months between Nov. 1957 and Sept. 1960 are reviewed. These cases were discovered in the course of 310 mastoidectomies and tympanoplasties performed in private practice. Sixty-six of these 310 operations were performed on children. None of these children was found to have tympanosclerosis.

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The 16 cases all had lesions within the middle ear cavity, with or without calcified plaques in the tympanic membrane. The case histories are correlated with the surgical and pathological findings. A rationale of management is discussed.

As yet there is not enough known about the disease, nor has there been a sufficient follow-up on surgical techniques, to permit final answers in this condition; therefore, this paper is presented as a preliminary, and it is hoped, provocative report, rather than a completed study.

REVIEW OF THE LITERATURE.

The concept of tympanosclerosis is not new. The earliest reference to this disease appeared in the German literature in 1869. At that time Von Troltsch[1] wrote:

"This sclerosis is a pathological process, in which the mucous membrane becomes denser, more rigid and inelastic. These changes impair the vibrating power of the membrana tympani very much, and of the membranes of the fenestra rotunda, and fenestra ovalis. They finally lead to a complete rigidity, calcareous or osseus degeneration of the membrane surrounding the stapes, ankylosis of the stapes, or of the membrane of the fenestra rotunda."

Schwartze[2] in "The Pathological Anatomy of the Ear" (1878) devoted a chapter to sclerosis of the mucosa of the tympanic cavity. At that time, however, no differentiation had been made between the condition and the bony changes of otosclerosis.

Walb,[3] writing in Schwartze's textbook, states:

"The scleroses consist of densely packed fibrous tissue with hyalin degeneration and with few nuclei. The sclerosis arises in the submucous layer, and there is a tendon-like alteration of the fibrous tissue in the deeper layers of the mucous membrane and the disappearance of many blood vessels. There are also fine granular changes in the mucous membrane with deposits of minute calcareous particles."

It is suggested that this is the otologic version of the healing phase of an inflammatory reaction comparable to the sugar coating thickening of the serous membranes covering the liver and spleen.

In "The Human Ear and Its Diseases" by Winslow[4] (1882) the changes in chronic inflammation of the tympanum are described. He writes:

"A thick fibrous membrane may cover the walls, obliterate the foramen rotundum, close the Eustachian tubes and antrum and even fill the whole middle ear with dense fibrous tissue. Like all such new pathologic formations, these are subject to fatty degeneration, sclerosis, contraction, calcification and ossification."

In 1883 Pomeroy[5] described the pathology of chronic aural catarrh quoting Schwartze:

"These membranes and bands undergo the same retrogressive changes that other similar tissue do, as atrophy, fatty degeneration, sclerosis and cicatricial contraction, calcification and ossification.

"The capability of vibration of the ossiculi is diminished by thickening or rigidity of the mucous membrane which covers these bones (sclerosis, calcification or ossification of the periosteal connective tissue with cellular and serous infiltration of the subepithelial layer) by synechiae and by the embedding of the bones in hypertrophied connective tissue."

In 1891 St. John Roosa[6] published "A Practical Treatise on Diseases of the Ear." Writing on chronic non-suppurative inflammations of the middle ear he described the pathology of proliferous inflammation as being made up of:

". . . connective tissue formations in the cavity of the tympanum . . . atrophy and fatty and fibrous degeneration in the tensor tympani . . . thickening and deposits of lime and of large round cells in the connective tissue stroma of the fenestra rotundum . . . pseudo membranous growths, sometimes filling the whole cavity with an irregular network

and sometimes covering the fenestra in the tympanic orifice of the Eustachian tube."

It is interesting to note that St. John Roosa makes no mention of the changes now recognized to be tympanosclerosis, in his chapter on consequences of chronic suppuration. He states that:

"The tympanum then will be converted into a dry chamber, its mucous membrane so altered that it scarcely secretes, and only on great provocation takes on inflammatory action."

His descriptions of lesions similar to tympanosclerosis appear only under chronic non-suppurative inflammations.

In 1894 Politzer[7] described pathological anatomical alterations under the heading "The Catarrhal Adhesive Processes in the Middle Ear." He wrote:

"The structural changes in the mucous membrane consist generally in partial or total transformation of the new formed round cells into fibrous connective tissue, interstitial hypertrophy of the mucous membrane, retrograde metamorphosis of the new formed tissue, shrinking sclerosis, atrophy and calcification."

Bruhl[8] in 1923 illustrated a tympanosclerotic lesion surrounding the stapes and ankylosing the ossicular chain under the heading of "catarrhus chronicus" of the tympanic cavity.

In 1930, Keeler[9] stated that:

"The sclerotic form of chronic middle ear catarrh shows hyperplasia instead of hypertrophy; in place of swollen and more or less edematous lining tissues, we find them hard, even fibrous in character with the blood vessels more or less sclerosed."

No reference was found in the literature to tympanosclerosis as such until 1955, when Zollner and Beck[10] wrote a paper on "Die Paukensklerose." This was the first time the term "tympanosclerosis" actually appeared. They stated that "Die Paukensklerose" caused middle ear deafness and histologically described the condition as:

"Hyalin degeneration of connective tissue with lime salt deposits in new bone formation."

They suggested it might be the result of an acute recurring otitis media in patients with an allergic constitution since eosinophiles were found. They described a series of nine cases which were operated upon.

Zollner[11] in 1955, writing in the *Journal of Laryngology*, on the "Principles of Plastic Surgery of the Sound Conducting Apparatus," again referred to tympanosclerosis in a case in which he demonstrated the surgical repositioning of the ossicles by rotating the incus.

In 1956 Zollner[12] published the most comprehensive article in the literature on tympanosclerosis. It included historical references, photographs of a lesion, a photograph of micropathology, and emphasized his recommendations for surgical management.

Shambaugh,[13] in 1959 in his text on surgery of the temporal bone, mentioned tympanosclerosis and refers to the articles by Zollner.

Goodhill,[14] in 1960, writing on "Pseudo-otosclerosis" included tympanosclerosis in his differential diagnosis. He mentioned it twice, giving case histories; once under lesions producing total ossicular chain fixation, and again in non-otosclerotic lesions of the stapes.

House and Sheehy,[15] in September 1960, published three case histories of tympanosclerosis, pointing out the differential diagnosis from cholesteatoma and otosclerosis and stressing proper surgical management for maximum hearing gain. In describing the pathogenesis of the lesion they state:

"The process takes place between the epithelium and the periosteum and destroys neither."

In describing surgical management they stated:

"The plaques can be safely removed (or left in place if desired) without fear of recurrence. . . ."

In summary, a review of the literature reveals detailed

descriptions of the pathological processes of tympanosclerosis as early as 1869. The condition was well described under the heading of catarrhal adhesive processes of the middle ear.

There was almost no mention of the condition during the first half of this century until the excellent articles of Zollner appeared in 1955 and 1956, setting forth the modern nomenclature and reviving the otologist's interest in "Die Paukensklerose."

DESCRIPTION OF THE LESION—PATHOLOGY.

Tympanosclerosis is being recognized more frequently due to the increasing use of the surgical microscope. The otosurgeon is finding this lesion to be not rare in the meticulous exploration of mastoid and tympanoplastic surgery. The pathologist will confirm the diagnosis, but only after the otosurgeon has recognized, removed and identified the lesion.

The pathogenesis and appearance of tympanosclerosis are beautifully described by Politzer[7] in his textbook:

"In cases where the exudative stage has not yet passed away, the hyperemic mucous membrane is usually unevenly tumefied, appears yellowish or bluish-red, infiltrated with serum, gelatinous, spongy, easily movable, uneven in surface, glandular, and shaggy. In consequence of this excessive proliferation of the mucous membrane, the depressions in the tympanic cavity, especially the niches of fenestrae ovalis and rotunda as well as the attic of the cavum tympani, are filled with succulent connective tissue, partially of new formation, which covers the head of the malleus and the body of the incus. Occasionally the mastoid antrum and mastoid cells are filled with masses of edematous connective tissue, or that inconstant fibrous network which extends from the malleus and incus into the mastoid antrum is thickened and hypertrophied. The mobility of the ossicula is impaired, but seldom quite lost by anchylosis at this stage. The increase in thickness of the lining membrane is caused partially by proliferation of the round cells, partially by new formed connective tissue. Therefore, by the round cells undergoing fatty degen-

eration, a partial repair of the mucous membrane may take place.

"In other cases where the secretion has totally ceased and where a complete transformation of the new formed tissue into connective tissue has already taken place, we find the mucous membrane generally smooth, several times as thick as usual, pale, of a dull, tendinous-gray colour, rigid, firmly united with its base, and only slightly movable. The condensation and rigidity affect not only the lining membrane of the tympanic cavity, but often also the folds of the mucous membrane and the ligaments which extend to the ossicula, as well as the coverings of the articula capsules. This thickening is generally most strongly marked where the ossicula touch the walls of the tympanic cavity. Only rarely partial calcification of the mucous membrane occurs and mostly on the promontory, as well as the hyperostotic narrowing of the cavum tympani."

In all of the cases described in this paper, the disease process has progressed into the connective tissue stage. There have been observed multiple very firm cartilage-like pearly white nodules measuring from 2 to 6 mm. implanted on the promontory and throughout the tympanic cavity. These may actually invade the otic capsule. Microscopic observation has also revealed generalized fibrosis and granulation tissue, multilocular and poorly circumscribed, involving the attic, and surrounding the ossicles producing fixation. Some of the most impressive lesions of tympanosclerosis have filled the entire aditus and attic areas forming a bed for, and completely immobilizing, the body of the incus. The gross appearance of these lesions has been that of smooth, firm, tough, cartilage-like tissue. Often the tissue occurs in onion-skin-like layers which can be separated. Grossly there appear to be two distinct types of lesions: *1.* one which can easily be dissected from its bed, leaving beneath an intact muco-periosteum, *2.* the other which extends not only through the mucosa but through the periosteum and burrows into the bone itself. Histologically the lesions appear similar except for the depth of penetration.

There are some lesions of tympanosclerosis which show epidermization suggestive of cholesteatoma formation and this has been confirmed microscopically.

In describing the histology of tympanosclerosis, Zollner[12] states:

"Our experiences . . . taught us two things: firstly, this type of sclerosis which in most cases constitutes a complete obstacle to vibration in the malleus and incus, and also in the stapes, can be removed without disturbing the ossicular chain. Secondly, that the sclerosis does not tend to recur after operation as it is a completed pathological process and in this it differs from the keloid changes in the skin."

Some of the cases in this series obeyed these rules and the tympanosclerosis could be dissected free from the ossicular chain; however, in numerous cases the ossicular chain was either completely enveloped by a massive block of tympanosclerosis or the tympanosclerosis was found embedded beneath the ossicular chain and could not be dissected free. Some tympanosclerotic lesions were of the invasive type and penetrated diffusely into the bony capsule. Other lesions had eroded away the fallopian canal and exposed the facial nerve. In several cases the lesion had invaded the stapedial footplate and appeared to penetrate into the vestibule.

It appears, therefore, that clinically there is not one, but two forms of the lesion: in the first form, there is an intact mucosa or mucoperiosteum present between the disease and the bone; in the second form, the disease penetrates mucosa and invades the bony capsule itself. Since it is logical and necessary that each of the two forms of tympanosclerosis be handled differently, it is suggested that they be differentiated by name. The superficial form which may be dissected free with relative ease, leaving behind an intact mucoperiosteum, may be referred to as *a.* sclerosing mucositis. The more destructive form which tends to respect no boundaries but invades and destroys bone might be referred to as *b.* osteoclastic mucoperiostitis.

The following histologic characteristics occur in typical

lesions of tympanosclerosis according to the consulting pathologist, Dr. Leo Weiss of Cedars of Lebanon Hospital, who has now studied the largest series of published cases:

1. Evidence of a chronic inflammatory process including granulation tissue, lymphocytes, plasma cells, and histocytes.

2. Evidence of dense fibrosis which may show hyalinization (*i.e.*, sclerosis).

3. Areas showing various stages of bone destruction, intertrabecular fibrosis and new bone formation.

4. Epithelium over the lesion may vary from columnar to keratinizing squamous epithelium. The latter appears to originate by metaplasia from the former and is the source of desquamating keratin, which retained in a loculated space is misnamed cholesteatoma.

5. Some lesions showed evidence of hemorrhage and hemosiderin containing granuloma derived from the hemorrhage.

6. Tympanosclerosis has been observed in the presence of an acute mastoiditis and/or "cholesteatoma".

DIFFERENTIAL DIAGNOSIS.

The typical patient in this series might be described as a middle aged male or female whose chief complaint is impaired hearing due to recurrent otitis media since childhood. Examination reveals the tympanic membrane of the impaired ear to have a sizeable dry marginal perforation. The remnants of tympanic membrane are usually abnormally thickened and may reveal calcareous deposits. Audiometry reveals a conductive loss of approximately 35 decibels. Mastoid X-rays reveal a sclerotic mastoid of infantile development with some evidence of attic erosion. If there are firm white nodular implants visible on the promontory, the diagnosis may be made by otoscopic examination. These nodules were visible in two of the 16 cases.

Thirteen of the 16 cases had perforated tympanic membranes. In three cases, the tympanic membranes were scarred

but intact. The majority of the series had perforated tympanic membranes with definite evidence of chronic recurrent suppurative otitis media. The primary differentiation must, therefore, be made between the tympanosclerotic lesion and a chronic mastoiditis with cholesteatoma. The otoroentgenologic diagnosis is usually cholesteatoma in both lesions. The differentiation is made in surgery by inspection and palpation. The tympanosclerotic mass is a firm fibro-sclerotic tissue with occasional areas of ossification. In contrast to this, the cholesteatoma sac is much less firm, and upon opening the sac the typical amorphous keratin debris is found. The final diagnostic separation is made by the pathologist. Both lesions may erode bone and produce injury to underlying structures such as the semicircular canals or the fallopian canal of the facial nerve.

The pyogenic granuloma so frequently encountered in mastoiditis is easily differentiated upon visualization at the time of surgery.

Certain specific types of chronic mastoiditis, such as that produced by tuberculosis, must also be differentiated from this lesion. Less frequently, eosinophilic or histocytic granulomas may simulate the chronic mastoiditis that is found with tympanosclerosis.

Three of our cases of tympanosclerosis had intact tympanic membranes. In all three the tympanic membranes were scarred and showed evidences of chronic infection. This smaller group must, therefore, be differentiated from other conductive lesions of tympanic origin. Here attention is called to the excellent differential diagnosis by Goodhill[14] in his paper on pseudo-otosclerosis, in which the differential diagnosis is made on an anatomical basis:

1. Total Ossicular Chain Fixation
   a. Chronic glue ear with adhesive fibrosis.
   b. Granulomatous otitis.
   c. Diffuse fibrosis.
   d. Tympanosclerosis.
   e. Pan-osteo-arthritis of ossicular chain.

2. Diseases of the Incus
   a. Traumatic dislocation of the incus.
   b. Fixed incus.
      i. incus annulus fusion.
      ii. incudo-malleolar fusion with cholesteatoma.
   c. Incus atrophy.
   d. Lenticular process necrosis.

3. Non-Otosclerotic Diseases of the Stapes
   a. Paget's disease.
   b. Osteogenesis imperfecta.
   c. Tympanosclerosis.
   d. Footplate arthritis.
   e. Peristapedial tent.
   f. Congenital fixation.

4. Anomalies of Ossicles and Windows

In addition to this, of course, there is true otosclerosis which will produce a conductive lesion and hearing impairment.

Tympanosclerosis has been discovered in approximately 5 per cent (16 out of 310) of the cases of chronic otitis media with mastoiditis which have come to surgery within the past three years. In many instances, the diagnosis has been suspected preoperatively; however, in only two instances, those in which the implants of tympanosclerosis could be visualized through a perforated tympanic membrane, was a definite diagnosis of tympanosclerosis made prior to surgery.

PRESENTATION OF CASES.

The following 16 case histories have been observed in the private practice of the writer and his two associates during the past 34 months. The chief complaint of the majority of patients was an impairment of hearing. The secondary complaint was otorrhea. All of these cases had diagnostic mastoid X-rays, and pre- and postoperative audiograms. They have been followed postoperatively from a minimum of one month to a maximum of 33 months.

History.

Case 1. J. L. This 49-year-old female patient was first seen ... office on September 11, 1945. At that time her chief complaint was that of impaired hearing bilaterally. The patient stated that her hearing had gotten progressively worse during the past nine years. It was further stated that the left tympanic membrane became perforated following scarlet fever in childhood and had drained intermittently since. Ti... ight tympanic membrane was perforated 14 years ago while swimm...

Physical Examination.

Examination in 1945 revealed the right ear canal to contain foul debris. The tympanic membrane was sharply retracted and showed a perforation in the posterior inferior quadrant. The left ear canal also contained foul suppuration. The left tympanic membrane showed a large posterior superior perforation which appeared to be marginal. Both the left and right Rinne tests were negative. The rhinolaryngological findings were not significant.

Roentgen examination revealed markedly contracted fully developed sclerotic mastoids bilaterally. The left mastoid showed evidence of attic erosion and probable cholesteatoma formation.

Audiometric Findings.

Audiometry revealed a bilateral conductive hearing loss with neural degeneration. The left speech reception threshold was 53 decibels and the left speech discrimination score was 92 per cent at +30 decibels.

Preoperative Impression.

The diagnosis at this time was bilateral otitis perforata with mastoiditis and probable left cholesteatoma. Surgical exploration was recommended.

Surgical Findings (Primary).

On November 19, 1957, the left ear was explored endaurally. The mastoid cortex was extremely thick and sclerotic. There was no pneumatization found. The antrum was clean. The aditus was filled with a firm white fibro-membranous lesion which appeared similar to cholesteatoma. The lesion had fixed the incus and filled the fossa incudus. When this firm white tissue was probed it was seen to be a fibro-calcific type of sac without any cholesteatoma content. The tympanic cavity was filled with this tissue. It had fixed the incudostapedial joint and malleus. The fibro-sclerotic lesion involved the promontory and completely involved both the horizontal and medial portions of the fallopian canal. The fibro-sclerotic lesions were removed as much as safety would permit.

Surgical Procedure (Primary).

The surgical procedure was a left endaural radical mastoidectomy. The tympanosclerotic lesion was partially removed.

Comment.

It was felt at this point that the disease had been incompletely removed and that further surgery on this ear should be performed. Approximately six weeks later on December 20, 1957, the ear cavity was dry. The patient was observed for routine hygiene of the ear cavity five times during the following year. It was then decided to re-explore the left ear with the hope of completely removing the disease and possibly doing a tympanoplasty. Audiometric re-examination of the left ear revealed a wide bone-air gap with fluctuation in bone conduction from previous studies. The

left speech reception threshold was 55 decibels and the left speech discrimination score was 86 per cent at +30 decibels.

Surgical Findings (Revision).

The left mastoid was re-explored postauricularly on March 26, 1959. The findings at surgery revealed the periantral mastoid bone had remained clear of disease. The horizontal semicircular canal showed a spotty erosion. The fallopian canal was densely covered by a firm white fibro-sclerotic material. This invaded the bony canal down to the facial nerve proper. It was impossible to dissect the lesion completely without risking harm to the nerve itself. The stapes was found to be fixed by a wedge of fibro-sclerotic material superiorly and anteriorly. The round window appeared to be normal. The Eustachian tube was moderately patent on exploration with a malleable probe. The fibro-sclerotic lesion completely surrounded the stapedial footplate. This mass was meticulously removed from the stapes, restoring mobility.

Surgical Procedure (Revision).

The surgical procedure consisted of a left postauricular radical mastoidectomy and a left tympanoplasty type III.

Pathology Report (Revision).

Two specimens were sent to the laboratory. The first specimen was a fragment of bone labeled incus. This was described microscopically as showing osseus necrosis and sclerosis with chondroid apposition. The second specimen was an irregular fragment of bony tissue measuring 0.5 x 0.3 x 0.1 cm. It was labeled tympanosclerosis and was described microscopically as showing sclerotic but viable bone with chondroid apposition. The microscopic diagnosis was sclerosing osteitis and ossifying sclerosis.

Postoperative Course.

Postoperatively the cavity healed well and was completely dry on April 21, 1959. There has been a mild improvement in hearing since surgery.

COMMENT.

This is the first case of tympanosclerosis which was identified and explored in this series. The ear was explored surgically on two occasions: first on November 18, 1957, and again on March 26, 1959. At both operations the tympanosclerotic material was well visualized and identified. The fibro-sclerotic lesions appeared to be grossly unchanged in the interval between the first and second procedures. There was no tendency for it to resolve itself spontaneously. The degree of progressive involvement which may have occurred was not grossly remarkable; therefore, it was felt safe to place a skin graft in the hopes of improving the hearing.

History.

Case 2. A. M. This 46-year-old male was first seen in our office on June 23, 1958. His chief complaint at that time was impaired hearing of at least

20 years' duration. He had been referred to our office from a city about 100 miles away in order to have stapes mobilization surgery to improve his hearing. It was stated that there had been repeated attacks of otitis media in childhood, which had eroded away both ear drums. A submucous resection of the nasal septum had been performed in 1931 and a tonsillectomy in 1938.

Physical Examination.

The entire pars tensa of the right tympanic membrane had been eroded away. A remnant of malleus was visible in the tympanic cavity. The middle ear was dry. There was no visible stapes. The round window was visible. The promontory was covered with firm white raised nodules. The right Rinne test was negative. The left tympanic membrane was eroded away posteriorly. The round window and the stapes were both visible through the perforation. The ear was dry. The left Rinne test was indefinite. The rhinolaryngological findings were not significant. Roentgen examination of the mastoids revealed infantile completely sclerotic mastoids, bilaterally.

Audiometric Findings.

Audiometry revealed a conductive loss bilaterally, more severe on the right. The right speech reception threshold was 40 decibels and the speech discrimination score was 98 per cent at +30 decibels. The left speech reception threshold was 30 decibels and the speech discrimination score was 97 per cent at +30 decibels.

Preoperative Impression.

The working diagnosis was bilateral otitis perforata with mastoiditis and probable right tympanosclerosis. An exploratory mastoidectomy and probable tympanoplasty on the right ear were recommended as the first procedure.

Surgical Findings (Right).

On December 11, 1958, the right ear was explored postauricularly. The mastoid cortex was found to be markedly sclerotic. The antrum was filled with a granuloma and what appeared to be a cholesteatoma which extended through the aditus into the tympanic cavity. This mass enveloped within its contents the body of the incus and the head of the malleus. Accordingly, the incus and malleus were removed. The firm white fibro-sclerotic tissue surrounded the stapes and had eroded the horizontal portion of the fallopian canal. The round window niche was normal. The Eustachian tube was normal. It was possible to remove completely the firm white tissue from the peristapedial area. The stapes was then mobile. A small amount of the fibro-sclerotic material remained at the genu of the facial nerve because it appeared to be intimately adherent to the perineurium of the nerve.

Surgical Procedure (Right).

A right postauricular radical mastoidectomy was performed. Following this, since the surgical area appeared to be safely clean, a full thickness skin graft was placed in contact with the stapes capitulum forming a Wullstein[16] Type III Tympanoplasty.

Pathology Report (Right).

Two specimens were sent to the laboratory for study. The first specimen was that of the incus and malleus. The microscopic description was sclerotic bone. The second specimen was the cartilaginoid tissue 0.3 x 0.3 x 0.2 cm. Microscopically, this tissue was partly lined by keratinizing squamous epithelium. Some of the keratin occupied a minute

cystic space. Beneath the epithelium were fused zones of granulation tissue, sclerosis, and dense bone. The pathological diagnosis was: *1.* sclerosis incus, malleus, *2.* tympanosclerosis, ear.

Comment.

The right cavity healed well, and the hearing improved so that it was at an equivalent speech reception threshold of 15 decibels at the end of April, 1959. It was decided at this time to explore the left ear and try to improve the hearing.

Surgical Findings (Left).

The left middle ear was explored via a tympanotomy incision and the incudostapedial joint was found to be intact. The incudomalleolar joint was also intact, and there was good transmission of motion from the malleus to the stapedial footplate. There was an excellent round window reflex. The Eustachian tube was patent. There were several 2 to 3 mm. whitish implantations over the fallopian canal and in the region of the round window niche and around the processus cochleariformis. The lesions were not confluent and appeared quiescent. A small opening was made into the antrum and there was no disease visible.

Surgical Procedure (Left).

A left postauricular tympanoplasty, type I, was performed after surgical exploration of the tympanic cavity and the opening of observation windows in the antrum and attic. The remnant of drum was de-epithelialized and a full thickness graft was placed over the manubrium and covered the entire widened posterior canal wall region.

Pathology Report (Left).

There were no specimens sent to the laboratory from this left ear.

Postoperative Course.

Postoperatively the left ear healed rapidly. One month later there was a left positive Rinne. Three months postoperatively the speech reception threshold in the left ear was at 10 decibels. This hearing gain has persisted.

COMMENT.

This case illustrates a bilateral involvement with tympanosclerosis. The right ear with the greater involvement requiring a Type III tympanoplasty has shown the poorer hearing result. It is likely that the tympanosclerotic process about the stapes has produced a partial refixation of the right stapes.

History.

Case 3. C. G. This 32-year-old female was first seen in our office on May 21, 1955. At that time her chief complaint was that of dizzy spells of many years' duration. The dizzy spells were described not as a true vertigo but more of a syncope with ataxia-like movements. These attacks had sometimes been accompanied by nausea and vomiting. There was a history of frequent earaches and left otorrhea throughout childhood. Many doctors had been visited for many and varied complaints, including gastro-intestinal symptoms and nervousness.

The patient was not seen again until four years later when she returned again complaining of dizzy spells. This time the dizzy spells were accompanied by tinnitus and an impairment of hearing in the left ear.

Physical Examination.

Physical examination revealed the right tympanic membrane to be intact, translucent and mobile. The left tympanic membrane had been replaced by a large neomembrane with a calcific plaque over the posterior inferior portion. The right and left Rinne tests were positive. The rhinolaryngeal findings were not significant.

Roentgen examination of the mastoids revealed limited pneumatization of the right temporal bone. The pneumatization of the left temporal

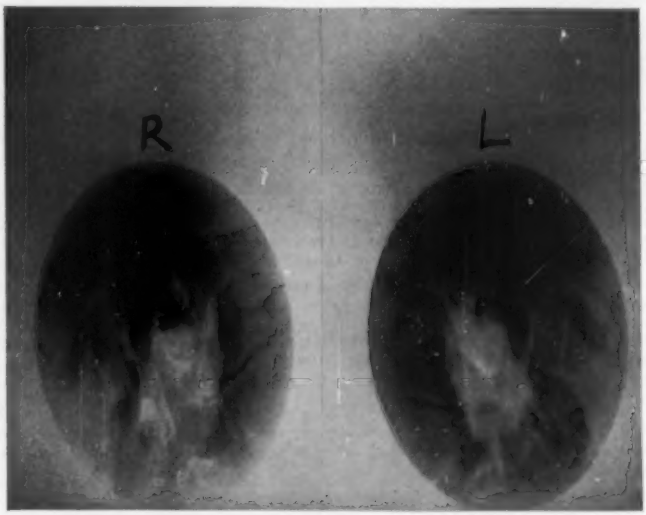


Fig. 1. Mastoid X-rays demonstrating left osteoclastic lesion simulating cholesteatoma in Case 3.

bone was limited to the left periantral area. There was a well defined area of bone destruction in the attic extending through a wide aditus into the antrum (see Fig. 1). The otoroentgenologic diagnosis was "chronic mastoiditis, left, with bone destruction in the attic, aditus and antrum, apparently a characteristic cholesteatoma cavity."

Audiometric Findings.

Audiometry performed on May 20, 1959, revealed a mixed type of loss in the left ear. The speech reception threshold in the left ear was 19 decibels and the speech discrimination score was 92 per cent at +30 decibels.

Preoperative Impression.

The preoperative impression was that of tympanic fibrosis with probable left cholesteatoma and labyrinthitis.

Surgical Findings.

On May 21, 1959, the left ear was explored endaurally. The findings at surgery revealed the antrum to be filled with a pyogenic vascular granuloma. The subincudal area was filled with a firm white fibrous mass with evidence of pressure erosion of the horizontal semicircular canal. A healed fistula was present. The peristapedial and intercrural regions were filled with a firm gray-white fibrous mass, but the footplate was mobile. There were tiny implants of the fibrous lesion throughout the entire tympanum.

Surgical Procedure.

A left endaural radical mastoidectomy was performed. The incus, malleus, and remnant of tympanic membrane were removed. The major areas of tympanosclerosis were removed. In view of the extent of the tympanosclerotic lesion, a skin graft was not laid over the remaining pathology.

Pathology Report.

The following four specimens were sent to the laboratory:

1. Tissue from the mastoid antrum which was microscopically described as cancellous bone with fatty marrow space heavily infiltrated with lymphocytes.

2. Calcified drum remnant which was grossly described as a 0.2 x 0.2 cm. yellow-white irregularly contoured piece of soft tissue. Microscopically this was described as focally calcified dense fibro-sclerotic tissue, partly covered by thin stratified squamous epithelium.

3. The incus and malleus were microscopically described as sclerotic ossicles showing focal resorption.

4. A spicule of red bone approximately 0.3 x 0.2 cm. was microscopically described as dense bone showing fibrous resorption.

Postoperative Course.

The left radical mastoid cavity was dry and well healed on April 25, 1960. There was evidence of active disease in the mastoid cavity. The multiple subjective complaints of dizziness and occasional headaches persisted in diminished intensity.

COMMENT.

This case illustrates the invasive potential of tympanosclerosis, producing a semicircular canal fistula and vertigo. The mastoid cavity shall be observed periodically. No further surgery is planned on this patient.

History.

Case 4. J. K. This 43-year-old female was first seen on August 31, 1959, at which time she had the complaint of right otalgia and otorrhea. She stated that this was an acute flareup of a chronic right ear infection which had been present since childhood. The otorrhea had been persistent, requiring the care of an otolaryngologist for the past ten years.

During these years aural polyps had been removed twice. Radium therapy had been applied to the area of the polyps. An adenotonsillectomy had been performed at the age of 34.

Physical Examination.

Examination revealed the right ear canal to be markedly inflamed with an area of bare bone visible on the posterior inferior canal wall. A large edematous aural polyp filled the remainder of the ear canal. The canal skin was inflamed and edematous. The middle ear was actively suppurating. The right Rinne test was negative. The left tympanic membrane was partially replaced by a neomembrane. It was intact and translucent. The left Rinne response was indefinite. The rhinolaryngological findings were not significant.

Roentgen examination of the mastoids revealed a well pneumatized left mastoid process. The right mastoid was poorly pneumatized and was of the juvenile type, with sclerosis and diffuse haziness consistent with an acute mastoiditis.

Audiometric Findings.

Audiometry revealed a 30 decibel conductive loss in the right ear, with normal hearing on the left. The speech reception threshold in the right ear was 24 decibels and the speech discrimination score was 94 per cent at +30 decibels.

Preoperative Impression.

The diagnosis at this time was of an acute exacerbation of a chronic mastoiditis with aural polyp. Prompt surgical treatment of this disease was advised.

Surgical Findings.

On September 3, 1959, the right ear was explored postauricularly. At surgery it was found that the cortex of the posterior ear canal had been eroded through by an acute osteomyelitis. There was a 1 cm. necrotic sequestrum of bone in the posterior canal area. There were few sclerotic mastoid cells. The attic and antral areas were filled with infected granulation tissue. The peristapedial area was invaded by a firm white fibro-sclerotic mass.

Surgical Procedure.

A right radical mastoidectomy was performed. The remnant of tympanic membrane and necrotic incus and malleus were removed. Due to the presence of the acute suppurative process, no tympanoplasty was attempted at this time.

Pathology Report.

The pathology in this case demonstrated both acute and chronic infection. There was a necrotic sequestrum of bone from the posterior canal area which was sent to the laboratory and described as an irregular fragment of brown bone 0.3 cm. in greatest diameter. The microscopic description was that of necrotic bone with leucocytes. Infected granulation tissue was removed from the attic and antral areas. Microscopically this had the appearance of fibrous tissue, bone and trapped glands.

The incus with surrounding tympanosclerosis was sent to the laboratory and was described as dense bone with marrow fibrosis and lymphocytes. The peristapedial white firm mass of tissue was described as fragments of pink fibrous tissue, the smallest being minute, the largest being 0.3 x 0.1 cm. The microscopic description was that of inflamed

fibrous tissue with trapped glands and hyalinized plaques. The deeper sections also showed calcification. Some segments had a tiny fragment of nerve (see Fig. 2).

Postoperative Course.

Postoperatively the mastoid cavity healed well, and at the end of one month was almost completely dry. On November 2, 1959, the cavity was completely dry. At this time the cavity showed a tendency to be filling in with fibrous tissue. On January 15, 1960, the mastoid bowl and the ear canal were completely filled with fibrous tissue, and the cavity was obliterated and dry.

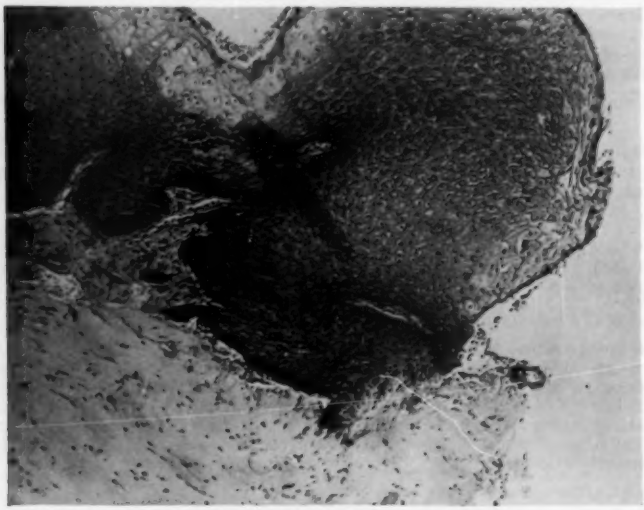


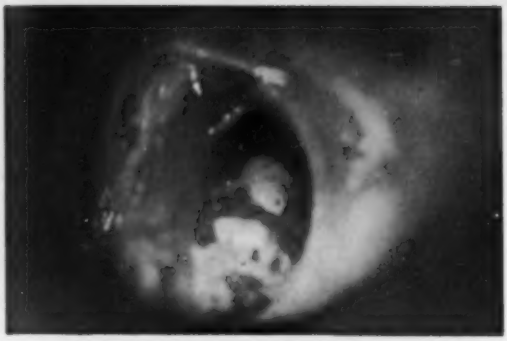
Fig. 2. Epidermatization over sclerosing fibrous tissue in Case 4. H&E x 33.

COMMENT.

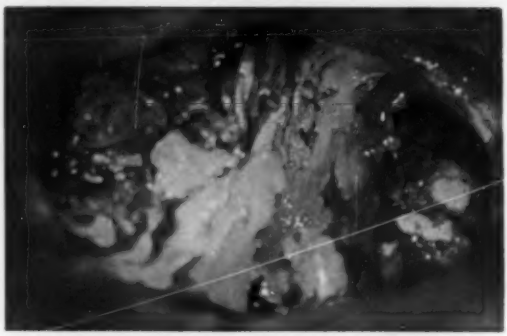
This case illustrates the presence of a tympanosclerotic lesion in the presence of an actively suppurating ear. The active suppuration prompted the surgical intervention and precluded any tympanoplastic repair at the time of surgery.

History.

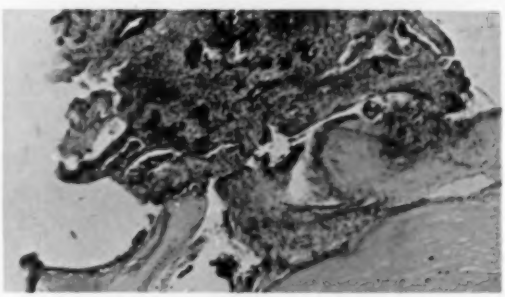
Case 5. D. C. This 55-year-old female was first seen in our office on May 16, 1959. The chief complaint at that time was impaired hearing of many years' duration. She stated that her difficulties had begun in



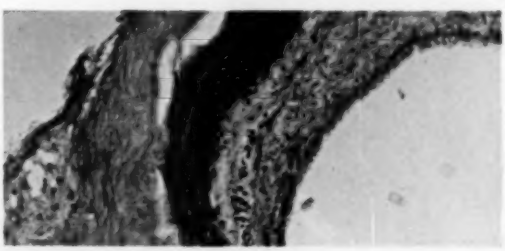
3. Tympanosclerotic implants on promontory as viewed through surgical microscope prior to operation in Case 5. 6X.



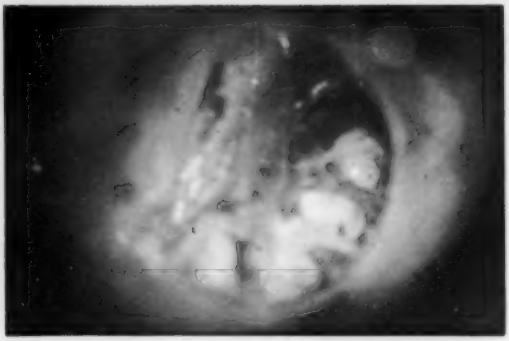
6. Extent of tympanosclerotic lesion after opening into the antrum in Case 5.



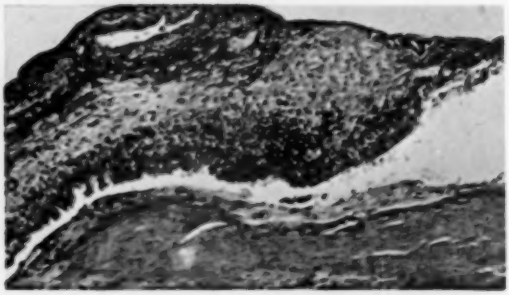
7. Granulation tissue (upper) hyalin sclerosis (lower right) and new bone trabeculum (lower left) in Case 5. H&E x 33.



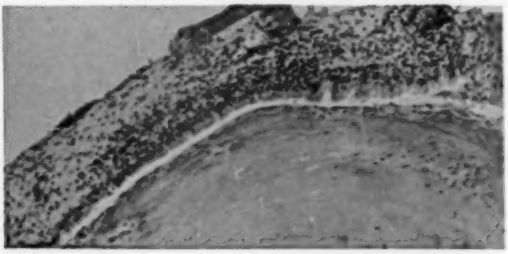
11. From left to right, successive layers of hyalin sclerosis (green), new bone (red) and granulation tissue in Case 11. Trichrome x 73.



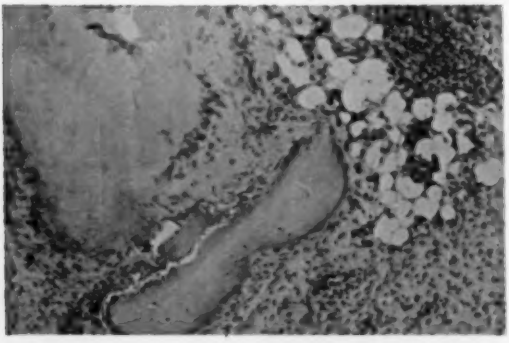
4. Tympanosclerotic implants on promontory as viewed through surgical microscope prior to operation in Case 5. 10X.



12. Columnar epithelium lines upper border of cleft. Inflammation and trapped glands in submucosa. Hyalin sclerosis in lower half, in Case 11. Trichrome x 73.



9. Midleft portion of Figure 8. Inflammatory infiltrate of columnar lined gland containing tissue above, hyalin sclerosis below with bone transformation to the right in Case 11. H&E x 73.



10. Right lower quadrant of Figure 8. Sharply defined areas of ossification in granulation tissue in Case 11. H&E x 73.

childhood with middle ear infections following scarlet fever, diphtheria and measles. There had been no otorrhea for the past 15 years. A hearing aid had been worn for several years. She further stated that there had been no otalgia, vertigo, nor recent otorrhea. An adenotonsillectomy had been performed in childhood.

Physical Examination.

Physical examination revealed the right tympanic membrane to be almost entirely eroded away except for a small area of Shrapnell's membrane. Over the promontory several implants approximately 3 to 4 mm. in size of firm white glistening tissue were visible (see colored plate Figs. 3 and 4). These appeared to be raised above the surface. The middle ear was dry. The left Rinne test was negative. The rhinopharyngeal findings were within normal limits.

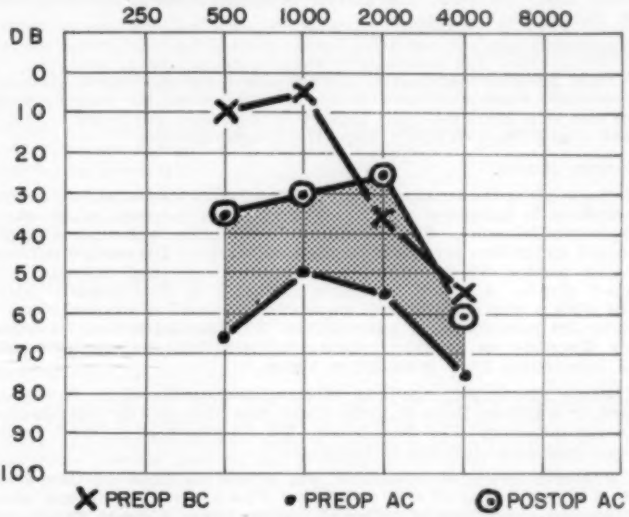


Fig. 5. Audiogram of the right ear showing preoperative air conduction and bone conduction, and postoperative air conduction in Case 5.

Roentgen examination revealed the left mastoid to be diploic in nature with evidence of attic sclerosis. The right mastoid was diploic, with an extensive attic erosion.

Audiometric Findings.

Audiometry revealed a marked conductive loss, bilaterally, with a high tone nerve drop (see Fig. 5). The right speech reception threshold was 60 decibels and the speech discrimination score was 96 per cent at +25 decibels.

Preoperative Impression.

The preoperative diagnosis was: *1.* right chronic otitis perforata with

mastoiditis and tympanosclerosis; 2. left adhesive otitis with tympanic fibrosis and probable ossicular discontinuity. Surgical exploration of the right ear was recommended, and a tympanoplastic repair was planned if possible.

Surgical Findings.

On September 3, 1959, the right ear was explored postauricularly. At surgery, the mastoid cells were found to be of the diploic sclerotic type. The malleus and incus were necrotic, and there was no evidence of the presence of the long process of the incus. The stapes was buried in a firm white mass of tissue which could be dissected free (see Fig. 6). The stapedial footplate was mottled in appearance. The stapes was intact and mobile. Beneath the ossicles and overlying the horizontal canal was a thick cartilaginoid layer of firm fibrous tissue. The bony fallopian canal had been eroded away by this process. The facial nerve was exposed above the genu. The antrum contained some of the same white firm fibro-sclerotic tissue.

Surgical Procedure.

A right postauricular radical mastoidectomy was performed. The tympanosclerotic mass was meticulously dissected from the stapes. A full thickness skin graft was then applied to the stapes producing a skin-to-stapes connection (Wullstein Type III Tympanoplasty).

Pathology Report.

A fragment of tissue approximately 0.5 cm. long by 0.1 cm. wide which appeared to be composed of white fibrous tissue was sent to the laboratory for examination. The microscopic description was that of hyperkeratotic epithelium overlying calcified and ossified fibro-sclerotic tissue. Another portion showed fused granulation tissue and sclerosis with trapped glands. A yellowish nodule composed of firm slightly friable tissue with a gritty cut surface measuring 0.5 x 0.3 x 0.1 cm. was also sent to the laboratory for examination. This specimen was microscopically described as showing sclerotic plaques joining bony trabeculae with intervening loose granulation tissue.

A single promontory specimen of tympanosclerosis which grossly appeared as a minute fleck of white tissue was also sent for examination. This was microscopically described by the laboratory as a tiny fragment without additional features to the above.

The fourth specimen submitted was a minute fleck of tissue, one surface of which was smooth and white. The microscopic description of this specimen was that of inflamed fibrous tissue, trapped glands, and sclerotic plaque with focal bone formation (see Fig. 7).

The microscopic diagnosis was osteitis ear, and sclerosis drum and ear.

Postoperative Course.

Postoperative examination on October 2, 1959, showed the right mastoid cavity to be well healed and dry. The right Rinne test was positive, and the hearing had improved. On April 30, 1960, the right cavity was well healed, and there was no evidence of recurrence of tympanosclerosis.

COMMENT.

In this patient the tympanosclerotic lesion was removed completely, and it appeared safe to apply a skin graft. This

has resulted in a dry ear and an improvement in hearing. Since this is now the better hearing ear, it is planned to do a surgical exploration of the other ear in the near future.

History.

Case 6. J. K. This 36-year-old male was first seen in our office on August 13, 1959. His chief complaint at that time was a recurrent right otorrhea since childhood. He stated that the tympanic membrane was ruptured by disease when he was quite young and that it became re-infected whenever he had an upper respiratory infection or got water in the ear. There had been no recent pain or otorrhea. His past history included a nasal submucous resection in 1950 and an adenotonsillectomy in childhood.

Physical Examination.

Physical examination revealed the right tympanic membrane to have a 4 mm. central dry perforation. The right Rinne test was negative. The left tympanic membrane was intact, translucent and mobile. The left Rinne test was positive. The rhinolaryngological findings were not significant.

Roentgen examination of the mastoids revealed a sclerotic, poorly developed right mastoid with some attic erosion.

Audiometric Findings.

Audiometry revealed a mixed type of hearing loss in the right ear, predominantly conductive. The right speech reception threshold was 42 decibels, and the speech discrimination score was 96 per cent at +25 decibels.

Preoperative Impression.

The diagnosis at this time was a right otitis perforata with sclerotic mastoiditis. Surgical exploration with the intention of performing a tympanoplastic repair was advised.

Surgical Findings.

On September 14, 1959, the right ear was explored postauricularly. The findings at surgery included a mastoid antrum which was filled with granulation tissue and also an extensive multilocular diffuse firm gray-white fibrous lesion. The primary location of this cartilaginoid mass was in the aditus region and extended into the middle ear. The incus and malleus were sequestrated and necrotic. The tympanic membrane was fibrotic. The gray-white fibro-sclerotic lesions extended through the otic capsule in lamellar fashion with layers of epithelioid tissue alternating with layers of bone. Upon removal one could almost see the endosteal membrane of the cochlea as well as the endosteal membrane of the horizontal semicircular canal. This very extensive involvement made it virtually impossible to do a complete removal. There was some extension of this fibro-sclerotic mass into the region of the horizontal semicircular canal and the peristapedial region. The mastoid bowl was clean.

Surgical Procedure.

A radical mastoidectomy was performed. In view of the extensive tympanosclerosis, a tympanoplasty was considered inadvisable at this time. A full thickness skin graft was used to line the mastoid bowl only.

Pathology Report.

Two specimens were sent to the laboratory, the first being the incus

and malleus from the right mastoid. They were described microscopically as ossicular bone fragments with a strip of surface squamous epithelium. The second specimen was a strip of whitish translucent membrane 0.5 x 0.2 cm. and seven minute fragments of similar material. The microscopic description was of small fragments of sclerotic hyaline tissue and bone fragments.

Postoperative Course.

Postoperatively the mastoid bowl healed slowly but was completely dry on January 9, 1960. Following an upper respiratory infection in March, the right mastoid cavity became moist and developed granulation tissue which has persisted until the time of this writing. There is a firm 4 to 5 mm. scar in the middle ear with satellite small white areas suggestive of the white fibro-sclerotic lesions found at surgery. There appears to be some Eustachian tube drainage.

COMMENT.

This case represents an extensive invasive tympanosclerotic lesion in a patient who has had chronic recurrent suppurative otitis media. No further ear surgery is planned at this time.

History.

Case 7. B. L. This 39-year-old female was first seen in our office on September 15, 1959. At that time her chief complaint was that of impaired hearing bilaterally. She stated that there had been recurrent attacks of otitis media with otorrhea and hearing impairment since childhood. She had worn a hearing aid when she was 18 years of age. Her past history included five surgical procedures for the removal of tonsils and adenoid tissue. She stated that she suffered from hay fever and bronchial asthma.

Physical Examination.

Examination revealed the right tympanic membrane to be intact, calcified, fibrotic, adherent and dry. The right Rinne test was negative. The left tympanic membrane showed a dry posterior superior perforation. There were areas of calcification and scarring with retraction and adhesions of the tympanic membrane. The left Rinne test was negative. The mucosa of the nasal turbinates was pale and edematous. The sinuses transilluminated well. The pharyngeal findings were not significant.

Roentgen examination of the mastoids in the Law, Stenvers, Towns, Myer and Owens modification revealed sclerotic mastoiditis bilaterally. There was no definite evidence of bone destruction. There was nothing to suggest abscess or cholesteatoma formation.

Audiometric Findings.

Audiometry revealed a bilateral conductive hearing loss. The speech reception threshold on the right was 51 decibels with a speech discrimination score of 98 per cent at +30 decibels. The speech reception threshold on the left was 32 decibels with 100 per cent speech discrimination score at +30 decibels.

Preoperative Impression.

The diagnosis at this time was that of bilateral chronic otitis media, associated with a bilateral chronic mastoiditis. Since the hearing was poorer in the right ear, an exploration of the right side was recommended as a primary procedure.

Surgical Findings.

On September 17, 1959, the right ear was explored postauricularly. The mastoid cortex was found to be sclerotic. The mastoid antrum and aditus were found to contain granulation tissue, together with an amorphous firm white fibro-sclerotic mass. The firm white cartilaginoid lesion was present in the tympanic cavity and involved the peristapedial and the peritubal regions. The incus and malleus were necrotic. The stapes was fixed because of intercrural and peristapedial fibro-sclerotic tissue. A partial stapedectomy was performed leaving the footplate. The crura and the fibro-sclerotic material were all removed. The stapedial footplate was left intact and mobile. The anterior crus remained and a polyethylene extension was placed upon this bony projection.

Surgical Procedure.

The procedure performed was a radical mastoidectomy. All visible evidence of tympanosclerosis was removed. A mobile stapes footplate was left and modified by the attachment of a polyethylene tube extension. A full thickness skin graft was then placed over the middle ear in contact with the polyethylene stapes producing a Wullstein Tympanoplasty Type III.

Pathology Report.

Several specimens were sent to the laboratory:

1. Several fragments of tissue composed of dense hyalin sclerosis with adjacent bone.

2. The incus and malleus composed of bone with cartilaginoid areas.

3. A fragment of brown irregular tissue measuring 0.2 x 0.1 x 0.1 cm. showed intermingled calcified hyaline fibrous tissue replacing bone. One space was lined by cuboidal cells.

The microscopic diagnosis was tympanosclerosis.

Postoperative Course.

The postoperative course was uneventful during the 19 days that the patient remained in Los Angeles. The cavity appeared to be healing well, and the patient returned to her doctor in Oregon. On December 24, 1959, a postoperative report was received from the Oregon doctor stating that the ear was completely dry and that the hearing was most markedly improved.

COMMENT.

This case illustrates the presence of tympanosclerosis in an ear that had been chronically infected for many years. The lesion could be removed completely at surgery, and a tympanoplasty was performed improving the hearing. Surgical exploration of the other ear is planned for the near future.

History.

Case 8. P. S. This 27-year-old female was first seen in our office on September 19, 1959. Her chief complaint at that time was of gradually progressive impaired hearing of the right ear. She stated that the right ear drum had been perforated at the age of nine months and that there had been recurrent otitis media since. The most recent recurrence of

otitis media was during the past year. Past history included an adenotonsillectomy performed in childhood.

Physical Examination.

The right tympanic membrane was perforated in the entire posterior quadrant. It was dry, and the incudostapedial joint was visible but appeared to lack continuity. The right Rinne test was negative. The left tympanic membrane was intact, translucent and mobile. The left Rinne test was positive. The rhinolaryngological findings were not significant.

Roentgen examination revealed the left mastoid to be well pneumatized and without evidence of cellular destruction. The right mastoid was smaller and diploic with moderate diffuse sclerosis.

Audiometric Findings.

Audiometry revealed a speech reception threshold of 42 decibels in the right ear. The speech discrimination score was 98 per cent at +33 decibels.

Preoperative Impression.

The diagnosis at this time was that of right otitis perforata, with sclerotic mastoiditis and probable ossicular necrosis. Accordingly, a right exploratory mastoidectomy and tympanoplasty was advised.

Surgical Findings.

On November 3, 1959, a right postauricular exploration was made. The lenticular process of the incus was found to be completely eroded and adherent to the head of the stapes by fibrous union only. The anterior portion of the tympanic membrane was four to five times normal thickness and calcified. The malleus was fixed by adhesive bands to the promontory. These adhesive bands were partially calcified. The round window was covered by scar tissue which was dissected free. The aditus and antral areas contained hyperplastic mucosa with some yellowish discoloration.

Surgical Procedure.

A postauricular mastoidectomy was performed. All of the fibro-sclerotic tissue was removed. The incus and malleus were removed. The calcified tympanic membrane remnant was removed. A full thickness skin graft was then laid over the middle ear in contact with the capitulum of the stapes forming a Wullstein Type III Tympanoplasty.

Pathology Report.

Several specimens were sent to the laboratory, including the calcified tympanic membrane remnant which was reported as showing hyaline sclerosis and ossification. There were also trapped glands present. The squamous epithelium was piled up in some areas. The ossicles were reported as showing an attached inflamed connective tissue, degenerative changes, and inflamed marrow spaces. The final pathological diagnosis was *1.* tympanosclerosis, ear; *2.* otitis; *3.* osteitis.

Postoperative Course.

The postoperative course was rather uneventful and on January 23, 1960, the right cavity was completely dry. Speech reception threshold at three months postoperatively was at 22½ decibels.

COMMENT.

This case illustrates a tympanosclerotic lesion which could

be easily removed and was suitable for tympanoplastic repair. The cavity has remained dry and the hearing improved.

History.

Case 9. L. V. This 36-year-old male was first seen in our office on September 11, 1958. His chief complaint at that time was of impaired hearing since infancy. He stated that he had had middle ear infections and had a bilateral myringotomy performed at the age of six months. Bilateral otorrhea had been recurrent up until high school age, but not since that time. An adenotonsillectomy had been performed in childhood. There had been a moderate amount of difficulty with chronic allergic rhinitis.

Physical Examination.

Examination revealed a large posterior superior perforation of the right tympanic membrane with visibly deformed ossicles suggestive of ossicular discontinuity. The right Rinne test was negative. The left tympanic membrane was scarred, intact with a neomembrane in the posterior superior quadrant. The left Rinne test was positive. The rhinolaryngological findings were not significant.

Roentgen examination of the mastoids revealed the left mastoid to be diploic in character, with evidence of periantral sclerosis and normal trabecular architecture. The right mastoid was infantile and undeveloped, completely sclerotic and revealed evidence of attic erosion.

Audiometric Findings.

Audiometry revealed a mixed type of hearing loss in the right ear and a predominantly nerve type of loss in the left ear. The right speech reception threshold was 40 decibels and the speech discrimination score was 100 per cent at +30 decibels.

Preoperative Impression.

The tentative diagnosis at this time was a right otitis perforata, with ossicular discontinuity and sclerotic mastoiditis. The second diagnosis was a left otitis adhesiva, with moderate neural degeneration. Covering prosthesis tests failed to reveal any improvement in hearing in the right ear. This suggested ossicular discontinuity, and the patient was lined up for an exploratory mastoidectomy and possible tympanoplasty.

Surgical Findings.

The mastoid cortex was markedly sclerotic. The antrum was found to be filled with a firm white fibro-sclerotic tissue which was present in layers which could be separated. This tissue completely enveloped the remnants of the ossicular chain. The incus and malleus were removed. Plaques of this fibro-sclerotic tissue extended superior to the horizontal semicircular canal and were removed. The disease also involved the fallopian canal in the peristapedial area and the processus cochleariformis. This material was meticulously removed from all areas including the intercrural space of the stapes. The stapes footplate was found to be rigid and, therefore, mobilized.

Surgical Procedure.

A right postauricular mastoidectomy was performed on December 4, 1958. The stapes which was found to be fixed, was mobilized, and then a full thickness skin graft was placed in contact with the stapedial capitulum producing a Wullstein Type III Tympanoplasty.

Pathology Report.

Specimens of the fibro-sclerotic tissue, together with the ossicles, were sent to the laboratory for examination. Microscopic description of the tissue was that it showed bone with fresh hemorrhage. There were also nodules of lamellated hyalinized collagenous tissue resembling free bodies or pseudo fibromas, as seen in hydrocele, joints, and mesothelial cavities. These nodules appeared to fuse with bony trabeculae. Pathological diagnosis was tympanosclerosis, ear.

Postoperative Course.

The right mastoid cavity was noted to have been healed and dry on February 11, 1959. The hearing was slightly poorer postoperatively. The Fletcher speech reception threshold was at 32½ decibels on March 19, 1959. On May 22, 1959, it was noted that a small perforation had developed in the anterior inferior position of the graft. This small perforation has persisted. The graft became adhesive to the medial wall of the promontory. Manipulation of the stapedial area through the graft produced vertigo on January 28, 1960. There was no visible evidence of further progression of the tympanosclerotic lesion.

COMMENT.

This case illustrates the tympanosclerotic lesion invading the peristapedial area and producing stapes fixation. Manipulation of the stapes one year after mobilization revealed that the area was still movable and had not been refixed by the tympanosclerotic disease. It is interesting to note that the pathologist described the lesion as being nodules of lamellated hyalinized collagenous tissue resembling free bodies or pseudo fibromas.

History.

Case 10. R. S. This 52-year-old female patient was first seen on July 3, 1957. Her chief complaint at that time was impaired hearing and tinnitus bilaterally. She stated that left otorrhea had been present since early childhood. There had been only occasional flareups of right otitis media. An adenotonsillectomy had been performed in childhood. The patient had suffered from migraine headaches for many years and was in the habit of taking medication for this malady.

Physical Examination.

The right tympanic membrane showed a dry central perforation. The right Rinne test was negative. The left tympanic membrane had a 4 mm. central posterior perforation which was dry. The left Rinne test was negative. The rhinolaryngological findings were not significant.

Roentgen examination of the mastoids revealed infantile development of both mastoids, with periantral sclerosis.

Audiometric Findings.

Audiometry revealed a conductive lesion, bilaterally, more marked on the left. The left speech reception threshold was 50 decibels and the speech discrimination score was 92 per cent at +30 decibels.

Preoperative Impression.

The preoperative diagnosis was that of a bilateral chronic otitis perforata, with sclerotic mastoiditis. The patient was considered a candidate for tympanoplastic repair, and surgery of the left ear was recommended. The patient left California and did not return until the end of December, 1959. During this period she had had some local treatment to the right ear, and examination on December 22, 1959, revealed that the right tympanic membrane had sealed over with a neomembrane. The left tympanic membrane was unchanged. A left mastoidectomy and tympanoplasty was recommended.

Surgical Findings.

The left ear was explored postauricularly on January 5, 1960. The findings at surgery included a densely sclerotic mastoid with a few diploic cells. The mastoid antrum and the small periantral cells were filled with a pyogenic granuloma. The incus and malleus were embedded in similar pyogenic granuloma. The ossicles were necrotic. Upon removal of the granulomatous sac, together with the ossicles, a firm gray-white fibro-sclerotic lesion was seen to involve the peristapedial area and its bordering fallopian canal in both the horizontal and descending portions. The invasion of the stapes, crura and footplate was so complete that a total stapedectomy was necessary in order to remove the lesion. The round window niche was found to be covered by a plaque of fibro-sclerotic tissue. The mucosa of the tympanic cavity was hypertrophied but intact. The Eustachian tube orifice appeared to be patent. A vein graft was placed over the naked oval window after which a good round window reflex was obtained.

Surgical Procedure.

A left postauricular radical mastoidectomy and total stapedectomy was performed. Following this a vein graft was placed over the naked oval window and a polyethylene strut placed atop the vein graft and beneath a full thickness skin graft converting this to a type III prosthetic tympanoplasty.

Pathology Report.

Six separate specimens were submitted. Granulation tissue from the aditus and antrum showed cholesterol granuloma. Similar cholesterol granuloma and nonspecific granulation tissue were noted in bone from incus and malleus. Portions from the stapes appearing grossly tympanosclerotic showed hyaline sclerosis replacing necrotic appearing bone. Diagnosis was: *1.* cholesterol granuloma; *2.* osteitis; and *3.* tympanosclerosis.

Postoperative Course.

The postoperative left mastoid cavity healed well and was noted to be dry on February 22, 1960. Air could be introduced beneath the graft by a Politzer maneuver with ease; however, there was no gain in hearing in the left ear. The speech reception threshold five months after surgery was at 42½ decibels.

COMMENT.

This case represents the co-existence of a cholesterol granuloma and tympanosclerosis in the same ear. The tympanosclerosis was of the invasive type and it necessitated a com-

plete stapedectomy in order to eradicate the disease and to attempt to improve the hearing.

History.

Case 11. J. M. This 20-year-old male was first seen in our office on July 15, 1959. The chief complaint at that time was of recurrent bilateral ear infections from the age of three. There had been recurrent left otorrhea for the past 17 years. There had been somewhat less difficulty with the right ear, although right otalgia and right otorrhea had also been recurrent. Impairment of hearing, more marked on the left, had been noted for many years. Past history included that of a tonsillectomy performed at the age of 12.

Physical Examination.

The left tympanic membrane had been destroyed by disease. The promontory was covered with moist granulation tissue, and there was mucopus present in the middle ear and ear canal. No ossicles were visible. The left Rinne test was negative. The pars tensa of the right tympanic membrane had been eroded away. Shrapnell's area was scarred but present. The tympanic cavity was dry. The right Rinne test was negative. The rhinolaryngological findings were not significant.

Roentgen examination showed the left mastoid cells to be diploic in type, with periantral sclerosis and evidence of attic erosion. The right mastoid cells were diploic, poorly developed, with evidence of periantral sclerosis and erosion in the attic area.

Audiometric Findings.

Audiometry revealed a conductive hearing loss, bilaterally, with good nerve function and a more marked impairment on the left. The left speech reception threshold was 65 decibels and the speech discrimination score was 94 per cent at +30 decibels.

Preoperative Impression.

The diagnosis at this time was that of: *1.* bilateral otitis perforata, suppurative on the left; *2.* left mastoiditis with probable cholesteatoma. Surgical exploration of the left ear was recommended as the initial approach.

Surgical Findings (Left).

On August 12, 1959, the left ear was explored postauricularly. The findings at surgery included a contracted sclerotic mastoid cortex. On entering the antrum a large cholesteatoma was seen filling the entire antrum and extending through the aditus into the attic area. The tegmen mastoidi had been eroded by the cholesteatoma and had exposed a 1 cm. area of dura. The dura was covered with granulations. Beneath the granulations the dura appeared compressible and intact. The malleus, incus and capitulum of the stapes were necrotic. The stapes footplate was intact and mobile. The mucosa over the promontory was intact. The round window niche was normal. The Eustachian tube orifice was patent. There was no evidence of the presence of tympanosclerosis.

Surgical Procedure (Left).

A left postauricular radical mastoidectomy was performed. In order to give sufficient projection to the crura of the stapes, a silver clip was placed on the anterior crural remnant and a polyethylene tube on the posterior crural remnant. A full thickness skin graft was then placed in contact with the stapedial projections producing a Wullstein Type III Tympanoplasty.

Pathology Report (Left).

Pathological diagnosis was of keratin granuloma (cholesteatoma).

Postoperative Course (Left).

The left mastoid cavity healed well, and the graft remained viable. The hearing improved to a speech reception threshold of 30 decibels within a period of three months. At the end of five months the speech reception threshold (Fletcher Formula) was 17½ decibels. On March 14, 1960, it was decided to explore and attempt to repair the right ear. The preoperative right speech reception threshold was 28 decibels, and the speech discrimination score was 94 per cent at +30 decibels.

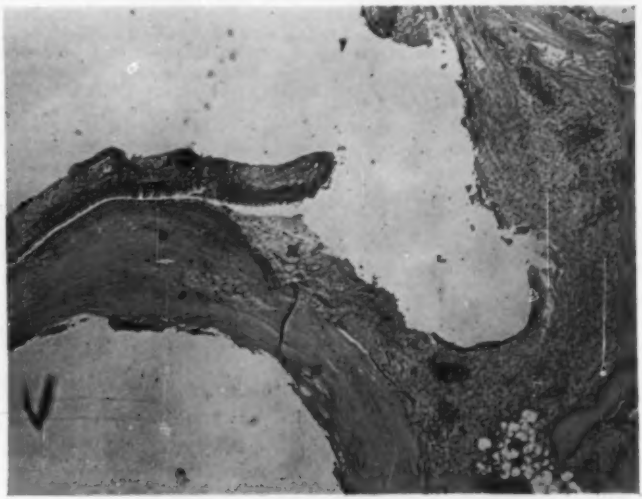


Fig. 8. Tympanosclerotic zone, tongue of inflamed mucosa and submucosa (to left) lies over sclerotic hyalin. Similar granulation tissue with focal collections of inflammatory cells in upper right corner. Ossification in lower left corner in Case 11. H&E x 33.

Surgical Findings (Right).

On March 15, 1960, the right ear was explored postauricularly. Since Shrapnell's area was intact an exploratory opening was made in the attic. This was found to contain granulation tissue and some firm white fibrosclerotic material. The mastoid antrum was then opened and also found to be filled with the firm white fibro-sclerotic tissue. The incus was found to be completely fixed. Upon removal of the remnant of malleus and incus a large plaque of cartilaginoid tissue was found to fill the entire aditus area, forming a bed for the body of the incus and producing complete fixation of the ossicular chain. Upon removal of this mass, the mucosa beneath appeared to be intact. The stapes was mobile and the round window reflex was readily obtained. The Eustachian tube orifice was open. The mucosa of the middle ear appeared to be intact.

Surgical Procedure (Right).

A right postauricular radical mastoidectomy was performed. Then a full thickness skin graft was placed in contact with the mobile stapes, forming a Type III tympanoplasty.

Pathology Report (Right).

Two specimens were sent to the laboratory, the first being a fragmented slightly firm whitish material measuring 0.7 x 0.5 x 0.2 cm. The second specimen was the incus and malleus. The microscopic description of the first specimen was that of fragmented material, partly lined by columnar epithelium of mastoid cell origin and composed of rather cellular fibrous tissue, vascular in some areas and avascular in others. There were focal collections of inflammatory cells, including polymorphonuclear leucocytes, lymphocytes, plasma cells, and siderophages. There was considerable often fibrillar calcification with hyaline osteoid zones. The osteo calcific areas were somewhat more strongly reactive with periodic acid Schiff than the collagenous areas. Trichrome and Van Gieson accent osteoid and bone by deep red staining. On the second specimen, the incus showed congestion and focal lymphocyte collections in fibrous marrow. There was some surface fibrous proliferation resembling some of the tympanosclerosis (see Fig. 8 and colored plate Figs. 9, 10, 11, and 12). The malleus was unremarkable. The diagnosis was tympanosclerosis.

It was most fortunate in this case to have had the pathologist present in surgery. He was thus able to visualize the ear lesion *in situ* and, therefore, better able to interpret the entire pathological picture. He examined the lesion through the surgical microscope before removal and under the microscope after processing the tissue.

Postoperative Course (Right).

Postoperatively the right cavity healed rapidly and was completely dry with a positive Rinne on June 9, 1960. Audiometry revealed a 15 decibel response at 500 cycles, but a drop down to 65 decibels at 1000 and 2000 cycles, giving a speech reception threshold (Fletcher Formula) of 40 decibels.

COMMENT.

This case illustrates a bilateral involvement with chronic otitis media and mastoiditis. In only one ear, however, was tympanosclerosis found to be present. The other ear was involved with cholesteatoma. It was valuable to have the pathologist present in surgery to correlate the surgical and pathological findings.

History.

Case 12. C. C. This 43-year-old male was first seen in our office on June 11, 1959. His chief complaint was of an impairment in hearing most of his life, becoming progressively worse during the past two years. His difficulty had started with repeated middle ear infections in childhood. There had been no recent otorrhea. An adenotonsillectomy had been performed at the age of five to six.

Physical Examination.

Initial examination revealed a dry perforation in the posterior inferior

quadrant of the right tympanic membrane. The right Rinne test was negative. The left tympanic membrane had a 3 mm. anterior inferior dry perforation. The rhinolaryngologic findings were within normal limits.

Roentgen studies of the mastoids (see Fig. 13) revealed poorly developed sclerotic mastoids with attic erosion.

Audiometric Findings.

Audiometry revealed a marked conductive deficit, bilaterally. Covering prostheses applied to both tympanic membrane perforations produced a slight improvement in hearing.

Fig. 13. X-ray of the right mastoid showing infantile sclerotic development with attic and antral erosion in Case 12. (Both cholesteatoma and tympanosclerosis found at surgery.)

Preoperative Impression.

A tentative diagnosis of bilateral otitis perforata with probable cholesteatoma and ossicular discontinuity was made. Surgical exploration was advised, with the intention of also doing a tympanoplastic repair if possible.

Surgical Findings.

On April 19, 1960, the right ear was explored postauricularly. The findings at surgery revealed the middle ear to be filled with a firm white fibro-sclerotic tissue which involved the promontory, the round window and the Eustachian tube orifice. The malleus and incus were buried in this lesion. The stapes was buried in an amorphous fibrous mass. Upon removal of the lesion it was found that the fallopian canal had been eroded in the area of the horizontal segment above the genu, exposing the facial nerve.

Surgical Procedure.

A postauricular right radical mastoidectomy was performed. The fibro-sclerotic tissue was meticulously removed leaving the stapes in place. The incus and malleus were removed with the diseased tissue. Because of the diffuse invasive nature of the lesion, there was no attempt at tympanoplastic repair at this time.

Pathology Report.

Six pieces of amorphous bony tissue were sent to the laboratory for examination. The specimens included three small pieces of dense bony ossicles, two of laminated keratin and attached granulation tissue and one tiny bone chip embedded in dense fibrous tissue. The microscopic description was reported as showing, in addition to unremarkable ossicles, dense hyaline sclerotic tissue overlying and fusing with osteoid tissue and pre-existent bone. The sclerosis is lamellated and whorled. Diagnosis was tympanosclerosis, ear.

Postoperative Course.

This patient was seen again on June 16, 1960, at which time it was noted that the cavity was healing well. Subsequent examination one month later revealed the cavity to be completely dry. Postoperatively the hearing was slightly poorer than previously.

COMMENT.

The tympanosclerotic lesion in this case involved all of the ossicles and extended through the fallopian canal exposing the facial nerve. Because of its invasive nature there was no attempt at tympanoplastic repair at this time. Present plans call for observation of the cavity over a period of time, with a possible tympanoplastic repair in the future. Since the second ear is relied upon for hearing and is asymptomatic at the present time, there are no immediate plans for surgery.

History.

Case 13. L. S. This 34-year-old male was first seen in our office on March 2, 1960. The chief complaint at that time was of impaired hearing in the left ear of many years' duration, becoming progressively worse in the past three months. There was a history of bilateral otorrhea since childhood. The hearing problem had been severe enough to warrant the use of an air conduction hearing aid in the right ear for the past nine years. An adenotonsillectomy had been performed in childhood. An automobile accident in 1950 had resulted in injury to the arm and face, but without any significant effect upon the ears.

Physico' Examination.

The pars tensa of the right tympanic membrane was completely absent. Shrapnell's area was fibrotic. The tympanic cavity was slightly moist. The mucosa was hyperplastic. There were no ossicles visible. The right Rinne test was negative. The pars tensa of the left tympanic membrane had been completely eroded away as well as part of Shrapnell's area. The remainder of the pars flaccida was thickened and scarred. There were no ossicles visible. The tympanic cavity was covered with mucopus. The left Rinne test was negative. The nasopharynx was moderately hyperemic and covered with a small amount of mucopus. The lingual tonsils were hypertrophied. The nasal septum was deviated to the right with partial low obstruction.

Roentgen examination of the mastoids revealed infantile development with marked sclerosis and attic erosion bilaterally.

Audiometric Findings.

Audiometry revealed a conductive hearing loss, bilaterally, more severe on the left. The left speech reception threshold was 36 decibels and the speech discrimination score was 92 per cent at +24 decibels.

Preoperative Impression.

The preoperative diagnosis was that of bilateral chronic otitis media with mastoiditis. Left mastoid exploration was recommended as the primary procedure.

Surgical Findings.

The left mastoid was explored postauricularly on April 25, 1960. The mastoid cortex was found to be sclerotic. The antrum was filled with a large pyogenic granuloma and what appeared to be a mass of cholesteatoma. The incus and malleus were found to be dislocated by a large gray-white fibro-sclerotic mass. The stapes was embedded in this same tissue. The round window was buried in adhesions. The Eustachian tube orifice was closed by granulations. The horizontal portion of the fallopian canal had been eroded by the cartilaginoid lesion, and the facial nerve was exposed.

Surgical Procedure.

A left postauricular radical mastoidectomy was performed. Because of the extensive involvement of the tympanosclerotic lesion and its invasive character, no attempt was made at tympanoplastic repair.

Pathology Report.

Two specimens were sent to the laboratory. The first was a firm mass of tissue 0.2 x 0.1 cm. which was described microscopically as a small fragment of calcified hyaline fibrous tissue attached to bone. The second specimen was the incus and malleus which were grossly recognizable and were described microscopically as sclerotic ossicles showing adherent fibrous tissue with some peripheral resorption. The diagnosis was: *1.* tympanosclerosis, and 2. sclerosis of the ossicles.

Postoperative Course.

The left mastoid cavity was noted to be completely dry on June 6, 1960. The hearing was poorer postoperatively with a speech reception threshold of 42½ decibels five months after surgery. A small 3 mm. plaque of firm white tissue was visible on the promontory of the left cavity on September 12, 1960.

COMMENT.

This case represents a diffuse invasive involvement of tympanosclerosis which precluded any attempt at tympanoplastic repair at this time. Since the right ear is the better hearing ear at present, no further surgery is planned for the immediate future.

History.

Case 14. E. M. This 34-year-old male was first seen in our office on May 19, 1960, complaining of impaired hearing in the left ear. He stated that he has suffered with recurrent bilateral ear infections most of his life. There had been a left otorrhea most recently.

Physical Examination.

Examination revealed the right tympanic membrane to have a 2 to 3 mm. central perforation in the anterior inferior quadrant. There was a calcified plaque over the posterior inferior quadrant. The right Rinne was positive. The left tympanic membrane was perforated superiorly in Shrapnell's area, and there was cholesteatomatous material visible. The pars tensa had been eroded by disease, and the middle ear was filled with granulation tissue. The rhinolaryngeal findings were not significant.

Roentgen examination of the mastoids revealed a well pneumatized right mastoid. The left mastoid was sclerotic with attic erosion and periantral sclerosis.

Audiometric Findings.

An audiogram taken on June 17, 1960, demonstrated a conductive deficit bilaterally. The left speech reception threshold was 38 decibels and the speech discrimination score was 100 per cent at +30 decibels.

Preoperative Impression.

The diagnosis at this time was that of a chronic purulent left otitis media with mastoiditis and cholesteatoma. It was recommended that a left exploratory mastoidectomy be performed.

Surgical Findings.

On June 20, 1960, the left ear was explored postauricularly. The mastoid cortex was found to be markedly sclerotic. Diploic mastoid cells filled with granulation tissue were removed. Cholesteatomatous matrix was found in the attic and aditus and removed. The incus was found to be necrotic. There was no evidence of a malleus. After removal of the surgical bridge a firm gray-white mass of sclerosing mucositis was found to involve the stapes, pyramidal eminence, and the horizontal portion of the fallopian canal. It invaded the cochleariform process and the bony capsule in such a way as to preclude complete removal. The stapes was mobile, and the round window niche could be visualized. A good round window reflex was obtained.

Surgical Procedure.

A postauricular left radical mastoidectomy was performed. No tympanoplasty was attempted.

Pathology Report.

Several specimens of tissue were sent to the laboratory, including the necrotic incus, cholesteatoma and tympanosclerotic fragments. The de-

Fig. 14. X-ray of the right mastoid demonstrating infantile sclerotic development with attic and antral erosion simulating presence of cholesteatoma in Case 15.

scription of the microscopic findings was that of "multiple fragments of sclerotic fibrous tissues, some containing tubular serous glands, some lined by keratotic stratified squamous epithelium; also cartilage presumably from the aditus." The second specimen was necrotic appearing bone.

Postoperative Course.

On September 9, 1960, the left mastoid cavity was dry. The hearing remained at the preoperative level.

COMMENT.

This case illustrates the co-existence of a tympanosclerotic lesion with cholesteatoma and a subacute inflammatory process.

History.

Case 15. E. D. This 40-year-old female was first seen in our office on April 18, 1960, at which time her chief complaint was that of impaired hearing, associated with recurrent tinnitus and otorrhea. Her past history included recurrent attacks of otitis media since infancy. It was stated that the ears had not been dry for longer than several weeks at a time. An adenotonsillectomy had been performed at the age of four.

Physical Examination.

The right tympanic membrane had been completely eroded away by disease. The middle ear was slightly moist, but there was no active suppuration. The ossicles were not visible. The right Rinne test was negative. The left tympanic membrane was completely eroded away except for a small area of Shrapnell's membrane. The left middle ear was slightly moist without active suppuration. No ossicles were visible. The left Rinne was negative. The rhinolaryngologic findings were not significant.

Roentgen examination revealed poorly developed infantile sclerotic mastoids (see Fig. 14) with evidence of attic erosion, bilaterally.

Audiometric Findings.

Audiometry revealed conductive losses, bilaterally. Audiometry was repeated, using Korojel tympanic prostheses resulting in a marked improvement in hearing, bilaterally. The right speech reception threshold was 27 decibels and the speech discrimination score was 96 per cent at +30 decibels.

Preoperative Impression.

The preoperative impression was that of a bilateral otitis perforata with tympanic fibrosis and mastoiditis. Since the hearing was improved so markedly using the Korojel prostheses, it was planned to do a surgical exploration and a tympanoplastic repair.

Surgical Findings.

On July 13, 1960, the right ear was explored postauricularly. At surgery the aditus and attic areas were found to be filled with hyperplastic mucosa and granulations. The malleus and long process of the incus were necrotic. There was diminished transmission to the stapes. The incus was found to be fixed and was removed. A 6 x 8 mm. firm white fibro-sclerotic mass of tissue was found to fill the entire bed of the incus. This entire mass was removed with relative ease, leaving the bone beneath bare of any mucosa. The bony wall of the lateral semicircular canal had been superficially eroded by the disease. Hyperplastic mucosa and granulation tissues filled the aditus, antrum and periantral cells. These granulations invaded the cells extending toward the petrous apex. All evidence of disease was meticulously removed. The Eustachian tube orifice was found to be patent. The round window was open, and a good round window reflex could be obtained.

Surgical Procedure.

A right postauricular radical mastoidectomy and a right tympanoplasty type III were performed. The tympanosclerotic mass was removed completely; therefore, a full thickness skin graft was placed in contact with the head of the stapes, producing a Wullstein Type III Tympanoplasty.

Pathology Report.

Two specimens were sent to the laboratory for examination:

1. A rather firm tissue 0.3 x 0.3 x 0.2 cm. labelled tympanosclerosis. The microscopic description was that of rather dense bone having a loose fibrous marrow. It was surrounded by dense fibro-sclerotic tissue with chronic inflammatory infiltrate, rare trapped glands and focal new bone formation.

2. The second specimen was that of the incus and malleus which were grossly recognizable. They were microscopically described as ossicles unremarkable except for fibrotic tissue attached to one.

The microscopic diagnosis was that of: *1.* tympanosclerosis; *2.* mastoiditis.

Postoperative Course.

The mastoid cavity healed rapidly, and four weeks postoperative on August 16, 1960, it was observed that the cavity was completely dry. The graft had taken completely, and the hearing improved to a satisfactory level.

COMMENT.

It was possible to remove completely the tympanosclerotic lesion and do a tympanoplastic repair in this case. The postoperative result thus far appears to be very good. The patient will be observed for any recurrence of disease. Surgical exploration and repair of the other ear will probably be undertaken in the near future.

History.

Case 16. H. H. This 64-year-old female was first seen in our office on June 10, 1959. Her chief complaint at that time was that of right otorrhea of many years' duration with an increase in severity during the past four weeks. The patient had had recurrent otitis media, following scarlet fever in childhood. A left radical mastoid operation had been performed 30 years previously with a resultant dry ear and poor hearing. The patient had worn a hearing aid in the right ear for the past ten to 12 years. An adenotonsillectomy had been performed many years previously.

Physical Examination.

The posterior half of the right tympanic membrane had been eroded away. Granulation tissue and cholesteatoma were visible in the area of the perforation. There was a pulsating suppurative discharge. The anterior half of the tympanic membrane was fibrotic and retracted. It was completely immobile. The right Rinne was negative. The left ear had been converted into a well healed dry radical mastoid cavity by previous surgery. The left Rinne test was negative. The rhinolaryngological findings were not significant.

Roentgen examination of the mastoids revealed a surgical defect of the left mastoid, consistent with a mastoid cavity. The right mastoid revealed infantile development with marked sclerosis and evidence of attic erosion.

Audiometric Findings.

Audiometry revealed a marked conductive deficit bilaterally, with evidence of neural degeneration. The right speech reception threshold

was 70 decibels and the speech discrimination score was 86 per cent at +25 decibels.

Preoperative Impression.

The diagnosis at this time was *1.* a right mastoiditis with granuloma and probable cholesteatoma; *2.* left well healed postoperative radical mastoid cavity. The ear was treated locally and responded by becoming dry and asymptomatic; therefore, the patient was observed periodically for the next year. In July of 1960, a flareup of right otorrhea and otalgia occurred. The patient was scheduled for a right mastoid exploration.

Surgical Findings.

On September 7, 1960, the right ear was explored postauricularly. The findings at surgery included a markedly sclerotic mastoid and a small antrum which was free of disease. The long process of the incus had become necrotic, and there was a discontinuity between the incus and stapes. The manubrium of the malleus had become fixed to the promontory. The remnant of tympanic membrane contained layers of calcified fibrous tissue and was completely immobile. The incus, malleus and remnant of tympanic membrane were removed. Atop the capitulum of the stapes was found a smooth cartilaginoid mass. This was removed. A 2 mm. area of the fallopian canal in its horizontal portion had been eroded by the disease. There was fibro-sclerotic tissue in the peristapedial area, and this was meticulously dissected resulting in a mobile stapes. A good round window reflex could be obtained. The Eustachian tube orifice was clean and open.

Surgical Procedure.

A right postauricular radical mastoidectomy was performed. Following this a full thickness skin graft was applied to the capitulum of the stapes forming a Type III tympanoplasty.

Pathology Report.

Several surgical specimens were sent to the laboratory including the malleus, incus, tympanic membrane remnants and fibro-sclerotic tissues. Microscopic examination revealed:

1. Sections of malleus and incus were not remarkable. The drum remnants showed residual tendonoid structure lined on one side by epidermis and on the other by columnar epithelium. Granular and diffuse calcification was present. The tympanosclerotic area was partly lined by squamous keratinizing epithelium and partly by columnar, but without actual demonstrated transition. Beneath the epithelium was visualized granulation tissue mingling with hyaline sclerosis, some of it calcified, and bone with fibrous intertrabecular tissue. Deeper fibrous tissue contained mucous filled glandular spaces.

2. A dense sclerotic plaque with focal dusty calcifications, partly lined by squamous epithelium and resembles the tympano-sclerotic plaque. No stapedial bone was recognized.

The diagnosis was: *1.* tympanosclerosis, ear; *2.* calcification, ear; *3.* no pathological diagnosis of ossicles, ear.

Postoperative Course.

Since this case is less than one month old at the time of this writing, there are no postoperative audiograms. The cavity appears to be healing well.

COMMENT.

This case illustrates tympanosclerosis involving the peristapedial area and erosion of the fallopian canal above the oval window. The major involvement, however, was in the tympanic membrane remnant.

Sixteen cases of tympanosclerosis, including nine females and seven males, have been reviewed. Of these 16 cases, 13 had perforations of the tympanic membrane, with 12 of the ears dry at the time of surgery (see Table I). All had impaired hearing; most had infantile sclerotic mastoids; all had a history of recurrent otitis media since early childhood. There were no instances of the disease having been found in children.

In view of these findings, it appears that tympanosclerosis is a definite pathological entity as a result of a degenerative process following long standing chronic suppurative middle ear disease.

The nature of the determining factor producing tympanosclerosis in some patients and not in others, following such a chronic suppurative process, has not as yet been discovered.

DISCUSSION.

It is interesting to note that the condition of otosclerosis was well known to the otologists as early as 1860. The lesions were precisely described by numerous authors under the headings of "Chronic Aural Catarrh," "Chronic Non-suppurative Inflammations of the Middle Ear," and "Chronic Inflammations of the Tympanum." The term, "tympanosclerosis" was first applied to this specific pathologic entity by Zollner and Beck[10] in 1955.

In our series of over 300 mastoid and tympanoplastic operations, we have discovered tympanosclerosis to occur in 5.2 per cent of the cases. Although 66 of these 310 cases were in ear diseases in children, none of the children demonstrated tympanosclerosis. This would appear to indicate that the process of tympanosclerosis requires a long period of

TABLE I.

Case No.	Age	Sex	Chief Complaint	Duration	Character of Tympanic Membrane	Perforation	Ear Wet or Dry	X-ray Findings	Pre-op SRT*	Procedure	Concomitant Ear Pathology	Post-op SRT*	Net Change SRT*	Result of Surgery
1.	50	F	impaired hearing	since childhood	anterior half completely calcified	intact	dry	infantile sclerotic attic erosion	52½	P-A Rad. Mastoid. Tympano. III		40	+12½	dry cavity
2.	46	M	impaired hearing	since childhood	eroded	right completely eroded	dry	infantile sclerotic	32½	P-A Rad. Mastoid. Tympano. III	pyogenic granuloma	20	+12½	dry cavity
3.	39	F	vertigo	since childhood	fibrotic neo-membrane, calcified plaque	intact tympanic membrane, neo-membrane	dry	infantile sclerotic attic erosion	12½	Endaural Rad. Mastoid.	pyogenic granuloma	32½	−20	dry cavity
4.	43	F	otalgia, otorrhea	since childhood	inflamed and edematous	obscured by aural polyp	wet	infantile sclerotic, acute antral clouding	25	P-A Rad. Mastoid.	pyogenic granuloma, sequestrated bone	27½	−2½	dry cavity
5.	55	F	impaired hearing	since childhood	fibrotic implants visible on promontory	7 mm. marginal	dry	diploic extensive attic erosion	52½	P-A Rad. Mastoid. Tympano. III	pyogenic granuloma	27½	+25	dry cavity
6.	37	M	impaired hearing, recurrent otorrhea	since childhood	fibrotic	4 mm. marginal	dry	infantile sclerotic attic erosion	37½	P-A Rad. Mastoid.	pyogenic granuloma	35	+2½	moist cavity
7.	39	F	impaired hearing	since childhood	fibrotic adhesive calcified plaque	intact	dry	sclerotic, no bone destruction visualized	45	P-A Rad. Mastoid. Tympano. III with prosthesis	pyogenic granuloma	10	+35	dry cavity
8.	27	F	impaired hearing	since early childhood	fibrotic	entire posterior half	dry	diploic moderate sclerosis	35	P-A Rad. Mastoid. Tympano. III		22½	+12½	dry cavity

TABLE I. (cont.)

Case No.	Age	Sex	Chief Complaint	Duration	Character of Tympanic Membrane	Perforation	Ear Wet or Dry	X-ray Findings	Pre-op SRT*	Procedure	Concomitant Ear Pathology	Post-op SRT*	Net Change SRT*	Result of Surgery
9.	36	M	impaired hearing	since early childhood	fibrotic	4-5 mm. posterior superior	dry	infantile sclerotic attic erosion	30	P-A Rad. Mastoid. Stapes Mobil., Tympano. III	pseudo fibromas	32½	–2½	dry cav. small perf. in graft
10.	55	F	impaired hearing, otorrhea, tinnitus	since early childhood	fibrotic	4-5 mm. central	slight suppuration	infantile sclerotic	40	P-A Rad. Mastoid. Tympano. III, vein graft prosthesis	cholesterol granuloma	45	–5	dry cavity
11.	20	M	impaired hearing	since childhood	fibrotic implants visible on promontory	6-7 mm. pars tensa	dry	diploic with attic erosion	22½	P-A Rad. Mastoid. Tympano. III	pyogenic granuloma	42½	–20	dry cavity
12.	54	M	impaired hearing	since childhood	fibrotic, decreased mobility	4 mm. posterior inferior	dry	sclerotic with attic erosion	40	P-A Rad. Mastoid.	cholesteatoma, pyogenic granuloma	50	–10	dry cavity
13.	35	M	impaired hearing	since early childhood	fibrotic Shrapnell's	total pars tensa	wet, serous	infantile sclerotic attic erosion	30	P-A Rad. Mastoid.	pyogenic granuloma	52½	–22½	dry cavity
14.	34	M	impaired hearing	since childhood	eroded	total pars tensa and part Shrapnell's	suppurating	sclerotic with attic erosion	35	P-A Rad. Mastoid.	cholesteatoma, pyogenic granuloma	35	0	dry cavity
15.	40	M	impaired hearing	since early childhood	fibrotic Shrapnell's	total pars tensa	dry	infantile sclerotic attic erosion	25	P-A Rad. Mastoid. Tympano. III	pyogenic granuloma	17½	+7½	dry cavity
16.	65	F	otorrhea, otalgia	since early childhood	fibro-calcific, immobile	entire posterior half	suppurating	infantile sclerotic attic erosion	62½	P-A Rad. Mastoid. Tympano. III	pyogenic granuloma	none		early healing

*SRT by Fletcher Formula.

time to develop—perhaps 15 to 20 years. This is in contradistinction to cholesteatoma, which is a not infrequent finding in chronic otitis media with mastoiditis in pediatric otology.

We were able to interest two very well qualified general pathologists in our problem: Doctors Leo Weiss and Nathan Friedman of Cedars of Lebanon Hospital. We were fortunate in having one of the pathologists, Doctor Weiss, present at the time of surgery so that he could view the lesion *in situ* through the surgical microscope and then further study the processed histopathological specimens. The Pathology Department of Cedars of Lebanon Hospital ran special stains to identify any characteristic type of cell structure or bacteria that might be identified with the tympanosclerotic lesion. No such specific characteristic was found. The presence of eosinophiles as mentioned by Zollner[12] did not appear to be consistent nor significant in these 16 cases, according to pathologist, Doctor Weiss. It was the impression of Doctor Weiss that the hyaline fibro-sclerotic lesion that was found and called tympanosclerosis was the end product of a non-specific degenerative inflammatory process.

It is interesting to note that the lesions in this series appeared to be of two separate types:

1. A sclerosing mucositis such as has been described by Zollner, which can be dissected cleanly with relative ease leaving an intact structure and mucosa or mucoperiosteum beneath it; the type of lesion which lends itself readily to surgical removal and invites tympanoplastic repair, and

2. A more destructive and invasive form of this disease, as demonstrated in the majority of our cases. In these cases the fibro-sclerotic lesions appeared to burrow through the mucosa and into the otic capsule of the tympanic cavity, destroying bone and invading soft tissue structures. For want of a better name, this invasive lesion has been termed an osteoclastic mucoperiostitis. In one case of this invasive type of tympanosclerosis, the patient came to surgery because of vertigo (see Case 3—C. G.). In several cases, the bony walls of the fallopian canal had been eroded away.

There were no cases of facial nerve paresis in our series; however, it was demonstrated in several cases that the tympanosclerotic lesion could invade and produce fixation of the stapes. In several cases the cartilaginoid lesions appeared to occur in layers which could be easily separated. Perhaps these layers of compression tissue are indicative of a time sequence.

It was interesting to note that in several cases there were epithelial elements found along with the tympanosclerotic lesion. In two cases (see Case 12—C. C., and Case 14—E. M.) cholesteatoma was actually present in the pathological specimen. It is a well accepted fact in otologic practice that no epithelial elements shall be buried under a skin graft in tympanoplasty. The likelihood of keratin granuloma (cholesteatoma) production is well accepted in this instance. Good otologic practice, therefore, contra-indicates the burying of tympanosclerosis with epithelial elements, just as it would of cholesteatoma.

In this series of cases ten were treated by surgical exenteration of disease plus tympanoplasty repair. In six cases a radical mastoidectomy without tympanoplasty was decided upon as the best course of action. These decisions were made on clinical judgment considering first the eradication of disease and the safety of the patient. As experiences increase it is hoped that a definite set of criteria can be established for advisability of tympanoplastic repair. It is accepted that epithelial lesions should not be buried beneath a skin graft. Does this also apply to the fibro-sclerotic tissue that is located in a non-critical area? Is the disease so slow growing that the patient might expect many years of improved hearing if a tympanoplastic repair were done, leaving some of this fibro-sclerotic tissue present? Is this disease a form of benign neoplasm?

One case (see Case 2—A. M.) had tympanosclerotic lesions bilaterally. Another patient (see Case 11—J. M.) had bilateral chronic otitis perforata with sclerotic mastoiditis, but only unilateral tympanosclerosis. What is the precipitating factor, or the triggering mechanism, for producing the typical

tympanosclerotic lesion in some patients with recurrent otitis media and not in others?

Time and continued study will perhaps answer these questions.

SUMMARY.

The long forgotten pathological entity of fibro-sclerotic cartilaginoid disease of the middle ear known as tympanosclerosis has been reviewed historically. The essential elements of a degenerative process of a chronic suppurative middle ear disease resulting in these deposits have been described and illustrated. It has been pointed out that there appears to be two separate types of this disease: *1.* a sclerosing mucositis which seems to be more superficial and amenable to complete surgical removal; *2.* an invasive form of osteoclastic mucoperiostitis which often defies complete removal.

Sixteen cases of tympanosclerosis were discovered in the course of 310 consecutive mastoidectomies. This represents an approximate 5 per cent incidence of this disease in overall middle ear disease and mastoiditis, which had come to surgery. Epithelial elements were found in some cases of tympanosclerosis and thereby signaled a warning against burying tympanosclerotic disease.

The final answers to the nature and management of this disease will come only from continued study and observation by teams of informed otologists and pathologists.

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A NEW METHOD OF PERFUSION FOR THE FIXATION OF TISSUES.*

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It is generally agreed that the best fixation and preservation of tissues for histological study is achieved by perfusion of the fixative solution through the circulatory system. The more common procedure of killing the animal, dissecting out the tissue or organ of interest, and immersing it in fixative gives only crude results and is unsuitable for the proper investigation of details of structure. After simple immersion the fixative can reach the interior cells of a piece of tissue only by diffusion, at a rate that may be of the order of a millimeter per hour or even less, and with a large block of tissue may require many hours for complete penetration. During this time the tissues are subjected to the dissociative actions of their self-contained enzymes, which destroy cell walls, digest the cytoplasmic contents, and generally reduce the tissue to an undifferentiated state.

Perfusion through the circulatory system rapidly brings the fixative in close contact with all the cells and depends to a minimum extent upon diffusion. The procedure is carried out on the anesthetized animal and begins with a flushing out of the blood by the injection of physiological saline solution, followed immediately by the injection of the fixative. When the injection is done properly, and only a brief time elapses between the surgical exposure and the passage of the fixative solution through the vessels, the tissues remain in

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a normal, living condition until they are killed by the fixative itself. They will then be transformed into a stable state that bears a relatively fixed relation to the normal and will safely withstand the further processes involved in the histological preparation.

An essential feature of this procedure is the speed with which it is carried out. The dissociative processes begin as soon as the tissues are deprived of oxygen, and they proceed at high rates until the fixative penetrates the cells in sufficient concentration to vitiate the internal enzymes; therefore, it is important to have a perfusion method that can be carried out quickly and that provides a free and copious flow of fluid through the blood vessels.

The conventional method of perfusion consists of inserting a cannula directly into one of the major arteries, such as the carotid artery, or of passing it through the left ventricle of the heart into the ascending aorta; then, after a vein is opened to provide an escape path, the injection fluids are introduced under a pressure that equals, or more often exceeds, the normal blood pressure of the animal. When performed by someone who has practiced it long enough to master all its details, this method yields excellent results. Even then, however, there are frequent failures, because something has gone wrong. Especially is this true in working with very small animals. With them it is difficult to make cannulas of the correct size, to insert them properly into the artery, and to retain them there with a ligature in the presence of the pressures required for the injection. When the vessels are particularly small, as they are in mice, bats, and birds, the cannula occludes the artery so seriously as to permit only a minute stream to flow.

A NEW PERFUSION METHOD.

The method of perfusion to be described here was designed to eliminate many of these difficulties. It has been developed for the study of the auditory structures of the mouse, and it will be described specifically for that application, but it can be adapted for any tissue or organ in any vertebrate

animal, large or small. It consists of an injection directly into the left ventricle of the heart, with all unused pathways tied or clamped off.

The principle of the method is portrayed in Figure 1, which indicates first the normal mammalian circulation, and then the path of flow of a fluid introduced into the left ventricle, with the right auricle opened and the unused vessels closed.

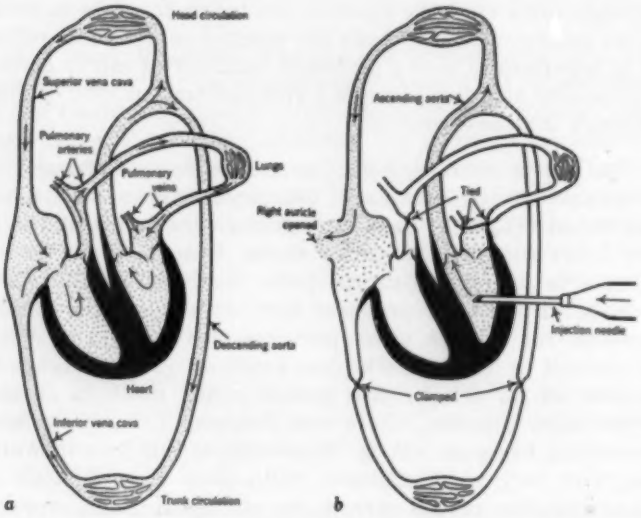


Fig. 1-a. Shows the normal mammalian circulation, in simplified and schematic form. The right pulmonary vessels have been removed. Only one superior vena cava is shown, though in the mouse there are two. b. Shows the routes of flow for perfusion of structures of the head. The injection needle is in the left ventricle.

It is essential to control the flow by positive closure of all undesired paths. The heart valves give no aid to this process, because they are made inoperative by the fixative.

A critical part of the equipment is a special injection head, shown in Figure 2. It consists of a central hypodermic needle, whose tip is adjusted to a length that is just sufficient to penetrate the wall of the left ventricle and to extend to the

middle of the ventricular cavity. This tip must not extend so far as to reach the medial septum, for then its opening will be occluded. For the adult mouse, the proper length is 2 mm., and a convenient gauge is No. 24. Enclosing this needle is a nosepiece in which an annular groove has been cut, and a second hypodermic needle enters this groove through an obliquely drilled hole. When the injection tip is pushed through the heart wall and the nosepiece is held in close

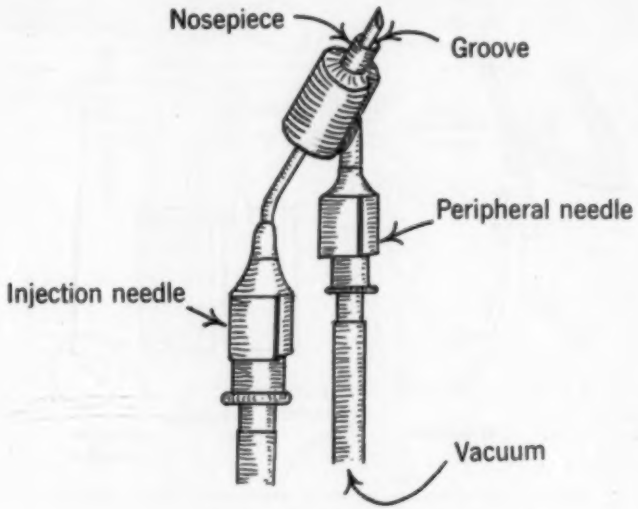


Fig. 2. The injection head.

contact with this wall with a moderate vacuum applied to the peripheral needle, the muscular wall is sucked into the groove. Then the nosepiece adheres to the wall and seals the puncture hole made by the central needle.

The complete injection apparatus is shown in Figure 3. The central needle is attached through a two-way stopcock to either of two fluid supply systems: one for physiological saline and the other for fixative solution. Between each container and the stopcock is a drip tube to catch any bubbles

coming through the supply tube. Care must be taken also to remove any bubbles between the drip tubes and the injection head, for if a bubble is allowed to enter the heart it can cause an air embolism somewhere in the circulatory system and block the further passage of the solutions. It is desirable to use transparent plastic tubing for all connections, both because this material is inert to the fixative solution and because a trapped bubble can easily be seen

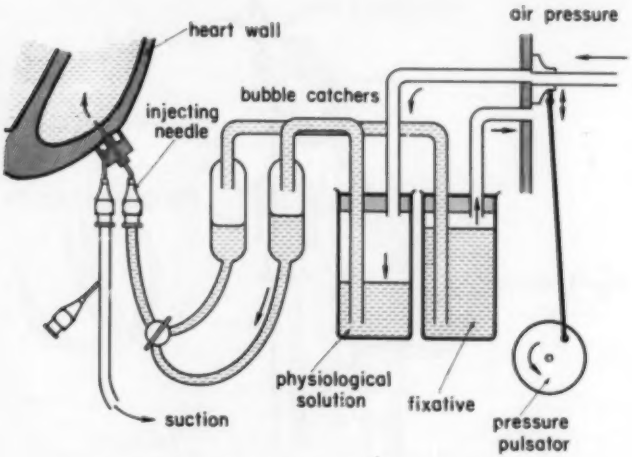


Fig. 3. The complete injection apparatus (not to scale).

through the walls. The connection tubes are made as short as possible and their bore is kept small—about 3 mm.

The physiological saline solution is used at a temperature of 40° C, and the fixative is at room temperature. We have used a commercial saline solution containing 5 per cent glucose, to which is added amyl nitrite in the amount of 1 cc per liter. The amyl nitrite is a vasodilator, and aids in the perfusion of the smaller vessels. The fixative may be any one of hundreds that have been developed. We have used one that contains potassium dichromate and, therefore, has an intense yellow color. The color is an advantage in judging

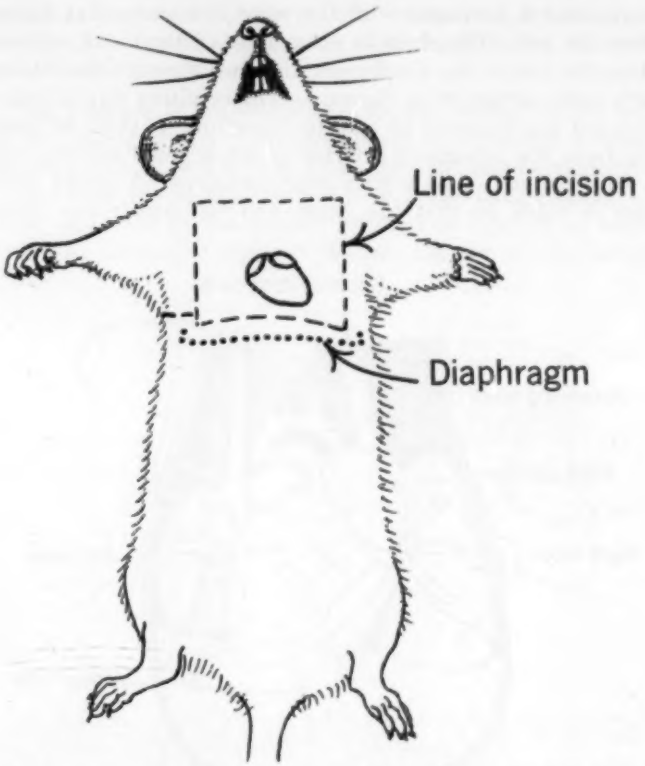


Fig. 4. The adult mouse, showing the location of the heart and guide lines for its exposure.

the success of the fixation; and if a colorless solution is chosen it is advisable to add to it a small quantity of dye to make its passage through the tissues evident. Most fixative solutions are highly corrosive to metals and fabrics, and produce deterioration of rubber. Special care must be taken to prevent contact with the skin and eyes of the operator.

The animal is anesthetized by placing it in a jar containing ether and about 0.25 cc. of amyl nitrite. It is then held firmly on its back on a platform that can be tilted to any desired

angle, and is arranged with the right side somewhat higher than the left. The chest is opened by cutting with scissors along the line of the diaphragm and then through the ribs on both sides, as shown in Figure 4. The resulting flap is pulled forward and removed by careful dissection. If there is bleeding from the mammary arteries or other small vessels, they are ligated or sealed with a thermocautery. A broad exposure is made so that the heart and its vessels are freely

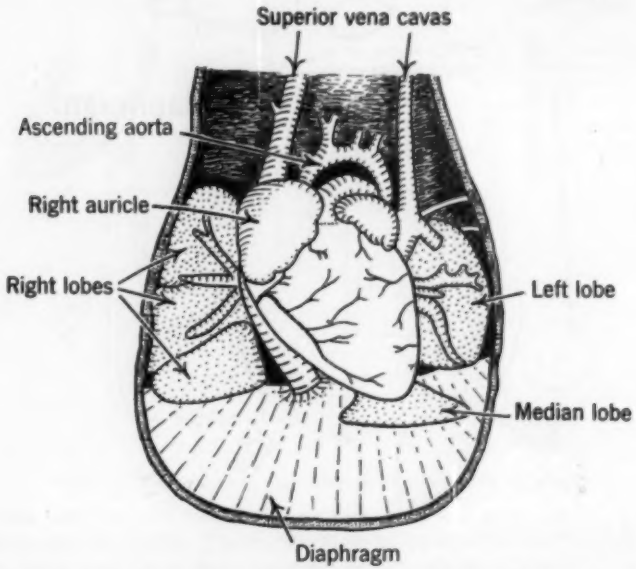


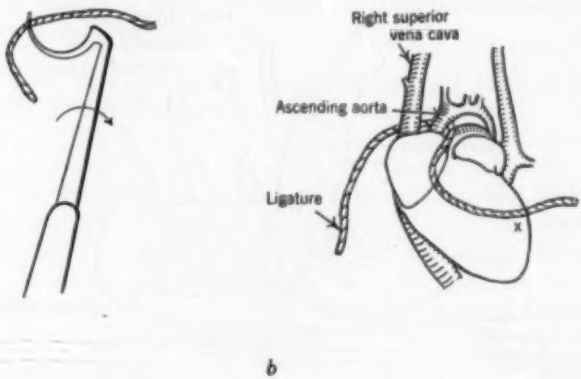
Fig. 5. View obtained after the exposure as described. Most of the pulmonary veins are seen, but the pulmonary arteries are behind the heart. The five lobes of the lungs are stippled.

exposed, and no retraction is required. The pericardium is removed, giving the view shown in Figure 5.

Often a mass of fat obscures the ascending aorta and the right superior vena cava, but it is not usually desirable to attempt its removal. If this mass is brushed forward with a pledget of cotton and the heart is stretched slightly by

pushing it posteriorly, these two vessels can be seen well enough for identification, and a single ligature can be passed beneath them.

The ligature is handled with a thread carrier of the form shown in Figure 6-a. This instrument is sometimes available commercially in sufficiently small sizes, or may be made from a fine steel needle that is held in a flame until red hot and bent while hot to the proper form. The ligature is passed from the operator's right to his left, the end of the ligature



a *b*

Fig. 6-a. The thread carrier. b. The ligature as passed beneath the ascending aorta and right superior vena cava. The point for entering the left ventricle (as described below) is indicated by "x".

seized with fine forceps, and the carrier then withdrawn. The ligature is finally pulled through, with the result as shown in Figure 6-b.

The heart must now be positioned, and this is done with a special manipulator consisting of a piece of 3 mm. plastic tubing that has been slightly heated at one end and this end flared. The tube is connected to a vacuum pump, and enough suction used to allow the flared end to adhere firmly to the heart wall but not to distort it unduly. Forceps must not be used to grasp the heart, as they cause injury. The tip of the heart is lifted and pulled forward between the two free

ends of the ligature, and the two ends of the ligature then are tied as in Figure 7, to include all the pulmonary arteries and veins, the inferior vena cava, and the left superior vena cava.

The right chest wall is divided almost to the spinal column and a hemostat applied to clamp the inferior vena cava and descending aorta. The heart is returned to its normal posi-

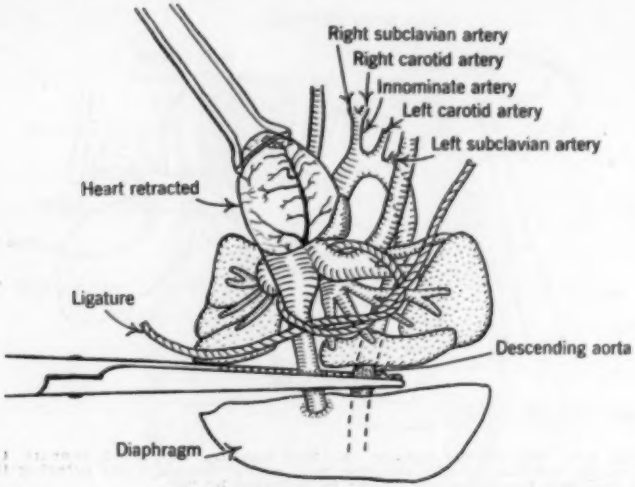


Fig. 7. View of the heart retracted forward, and the ligature formed into a loop to include the pulmonary vessels, inferior vena cava, and left superior vena cava. The hemostat is used to clamp the descending aorta, and it is usually convenient to include in its bite the inferior vena cava also, though this vessel has already been tied off.

tion, and then turned about 40° so that the left ventricle is nearly facing the operator. A wide tear is made in the wall of the right auricle to provide an escape path for blood and the perfusion fluids.

With the stopcock turned to connect the injection needle with the channel containing saline solution, the pressure is adjusted so that drops slowly appear at the injection tip. This is to prevent the formation of an air bubble at the tip.

The needle is then passed through the wall of the ventricle, with suction on the peripheral needle, and full pressure is immediately applied. The point of the puncture is indicated by "x" in Figure 6-b.

For this perfusion we have used pulsating pressure in imitation of the normal action of the heart. Such pressure is able to force fluid through minute vessels more successfully than a steady pressure when blood corpuscles are present. The pulsations move the corpuscles to and fro and break up any clumps that are formed. As soon as the flow is established, a holder is used to replace the operator's hand in supporting the injection head.

Care must be taken that a blood clot does not form in the right auricle. If this happens it is lifted out with fine forceps. The blood should flow out promptly, and the flushing should be continued until the escaping fluid is pale pink in color, and there are no longer any prominent red clouds. It is not desirable to continue until the fluid is entirely clear, for this takes so long as to expose the tissues to serious anoxia. A suction tube is used to remove the escaping fluids as rapidly as they appear, but its end is not applied directly to the auricle. The prompt removal of waste fluids reduces the escape of amyl nitrite fumes to the surrounding air. These fumes are disagreeable, cause headache in some persons, and are even dangerous if breathed in excessive amounts.

When the escaping fluid becomes pale pink in color, as described, the stopcock is turned to shut off the flow of saline solution and to start the flow of fixative. The perfusion of fixative is continued for 30 minutes or more, with refilling of the supply container if necessary. This container must not be allowed to become empty, for then air would be forced into the blood vessels. The pressure is removed and the stopcock turned to the "off" position during refilling.

When the perfusion is complete the injection apparatus is disconnected and promptly cleaned. The auditory bullas are exposed and opened, and note is taken of any signs of middle ear disease. The two temporal bones, including the entire

labyrinth and middle ear structures, are dissected out as a single piece of tissue. Fine saws are best for this removal, as they are less likely to cause fractures of the brittle bones than other instruments, such as rongeurs. The two temporal bones are left connected at the base because attempts to sever them may produce fractures that involve the labyrinth. They may safely be separated later after decalcification, but for these small animals we ordinarily do not separate them but simply process them as a pair. In removing the block of tissue we sever the VIIIth nerve at the internal auditory meatus and then clean out the brain tissue. We leave the cranial walls and shape them so that when the block is inverted, resting on these walls, the cochlear axis is horizontal. After the tissue is embedded in celloidin, the cranial surface is fastened to a fiber block for holding in the microtome, and the sectioning begins on the ventral surface. There is then a safe amount of space between the semicircular canals (which will be sectioned last) and the fiber block. This orientation gives a horizontal plane of sectioning, which is generally suitable for studies of the ear in all the mammals except the primates.

After removal as described, the block of tissue is placed in a bottle containing 50 times its volume of fresh fixative, and this solution is changed daily for one week, after which time the fixation will be complete.

PRECAUTIONS.

To insure success in this procedure it is necessary to take certain precautions. The necessity for speed in starting the initial flow of fluid has already been emphasized. The time from the opening of the chest to the beginning of the flow of saline solution should not exceed one minute. If it is longer the blood may coagulate in some of the lesser vessels, especially in the small and tortuous cochlear artery, so that the fixative fails to reach the ear.

The main bulk of the blood must be removed by flushing with saline solution, for failure to do so will leave enough in the small vessels to be coagulated by the fixative, thus

blocking the path to the ear. Some experience is necessary in judging the moment when the switch to the fixative solution should be made.

The injection pressure should be adjusted between 3 and 6 lbs. per square inch. The exact amount depends upon the size of the vessels, which varies among animals. When the pressure is correct, there will be a slight dripping of fluid from the nose, especially after the fixative begins to flow. A copious discharge from the nose or a prominent edema of subcutaneous tissues will indicate that the pressure is excessive.

It is best to introduce the holding device for the injection head before the fixative is injected. It may be applied later, however, if great care is taken not to disturb the preparation in doing so. As soon as the fixative reaches the blood vessels their walls become hard and brittle, and they are damaged by movements that ordinarily would be tolerated.

It is essential that the injection equipment, including all tubes and containers, be thoroughly cleaned after each use. The fixative solutions will corrode and clog the injection needles if retained in them for any considerable time; also all contamination of the saline solution must be avoided, for even a trace of fixative will produce coagulation of the blood at the first stage of the flushing process.

SIGNS OF GOOD FIXATION.

When the procedure is correct the solutions flow freely, and a copious amount of blood escapes in the first few seconds. Within ten seconds after the fixative begins to flow the skin of the face becomes noticeably yellow. The pinna also shows this color, as do the eyes of light-colored animals. When the fixation has continued a few minutes the fore part of the body becomes rigid.

When the fixation is complete the skin is strongly yellow. Dissection will show that all parts of the head are well colored, including all the muscles. The masseter muscle is a

particularly good indicator, for its fixation seems to occur last among all the muscles.

When the cranium is opened the condition of the brain is noted. All parts should be firm and well colored. If any portion remains soft and white there has been a blockage of some of the vessels of the head. When this condition is found, the tissues may as well be discarded at once, for any further processing of them will be wasted effort. It is our experience that any procedure that fails to give all the signs of perfect perfusion as described here will give sections in which the faulty fixation is painfully evident.

The posterior part of the body should not show the above changes. If they do, the descending aorta was not properly clamped. The trunk circulation then becomes a by-pass of low resistance, and the pressure to the head is correspondingly diminished; likewise, if the pulmonary vessels are not clamped, the lungs form a by-pass, again limiting the amount of fluid going to the head.

If this procedure is adapted to other tissues or organs, in a region of the body other than the head, the clamping pattern must be altered correspondingly, so as to include in the perfusion only the desired region. It is of course possible to fix the entire animal, but such fixation is inferior to what may be accomplished in a limited region. With small animals in particular the volume of fluid that may be forced through the aorta in a short time is too small to give optimal fixation of a large amount of tissue.

ADVANTAGES.

This procedure has a number of advantages over former methods of perfusion, such as passing a cannula through the left ventricle into the ascending aorta. The procedure requires less skill, and is more quickly mastered. There is no need to prepare a series of cannulas to meet the demands of varying sizes of vessels, and to work with small and delicate vessels. The frequent mistake of passing the cannula through the bicuspid valve into the left auricle instead of

through the aortic valve into the ascending aorta is avoided. Avoided also is the more serious accident of suddenly penetrating the aorta with the cannula while applying force in the effort to get past the aortic valve.

The procedure involves a minimum of disorder and muss. It does not even require a sink, but can be carried out anywhere in the laboratory.

Because the fluids are introduced into a relatively large chamber of the heart, and the aorta is not partially closed by a cannula, the flow is maximum, the blood is evacuated promptly, and the fixation is correspondingly rapid. The results, therefore, are more reliable and standardized.

SUMMARY.

A method was developed for the perfusion of a fixative solution directly into the left ventricle of the heart. The procedure is relatively easy to carry out, especially with small animals, and gives uniformly good results.

TEXAS UNIVERSITY ALLERGY COURSE.

The Post Graduate School of the University of Texas and The Baylor University College of Medicine will present a course in Allergy to be conducted by Herbert J. Rinkel, M.D., February 5-9, 1962, in Houston, Texas.

The course will be followed by a meeting of the Gulf Coast Allergy Study Group on February 9th and 10th.

For further information, write to: Office of the Dean, University of Texas Post Graduate School of Medicine, 410 Jesse Jones Library Building, Houston 25, Texas.

THE BING TEST IN THE DIAGNOSIS OF DEAFNESS.*

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INTRODUCTION.

In 1891 Albert Bing[1] published an article in the Wiener Medicinische Blatter with the title: "A new tuning fork test. A contribution to the differential diagnosis of the diseases of the conductive and perceptive hearing apparatus."

In that article he stated that if he held a tuning fork of middle frequency on the center of the head or on the mastoid process until it was not heard any more and then blocked the same ear canal lightly with a finger, the sound returned again, and it was heard for some time. He called the perception of this new sound "secondary perception" to differentiate it from the perception of the first sound, which he called "primary perception." He explained this phenomenon with Mach's theory of bone conduction. According to this theory, the vibratory energy is transferred to the cochlea from the vibrating head. Since "the sound conducting apparatus is well adapted for transmitting sound energy from the air to the labyrinth, according to the law of reciprocity, it is equally well adapted for transmitting energy in the opposite direction. An obturation of the meatus, which stops the drain, increases the level of kinetic energy in the labyrinth and increases bone conduction."[3,4]

He called the test positive, when the sound returned after blocking the ear canal, and negative, when the sound did not return.

If the positive test is a normal phenomenon, then the negative test is a pathological one. When the test is negative and all the other findings in the middle ear are negative, otoscle-

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rosis is the probable diagnosis. He finished his article with the hope that his colleagues would try his test, and would find it helpful among other tests in the differential diagnosis of hearing problems.

The purpose of this investigation was to determine the value of the Bing test in the diagnosis of deafness in general and especially in the differential diagnosis of conductive lesions, particularly between otosclerosis and other middle ear lesions.

The sound transmitted to the cochlea through the skull is called "bone conducted sound."[5,6] The threshold of bone conducted sound is considered the measure of the function of the cochlea. If an oscillator is applied to the mastoid area and the ear canal is open, the threshold of hearing is called the "relative bone conduction."[5,6] When the ear canal is blocked with a finger or with a piece of wet cotton, the threshold of hearing for low tones will be increased, and this threshold is called the "absolute bone conduction."[5,6] The difference between absolute and relative bone conduction is measured in the Bing test. It is positive when these two differ, and negative when they do not. It is a well known fact, from previous investigations, that the Bing test is positive in normal hearing ears and in perceptive deafness but is negative in otosclerosis and other lesions involving the conductive system.

METHOD OF INVESTIGATION.

More than 300 ears were examined as they were encountered in the private practice of this author and in the consultation service of the Ear, Nose and Throat Department of Bellevue Hospital. A complete history of these patients was taken and an ear, nose and throat examination was done. A clinical diagnosis was established, then an audiometric test was made by the writer to assure the same technique throughout the series. The hearing tests were made in a sound-treated room with an ambient noise level not exceeding 35 db sound pressure level. A Maico E-2 audiometer was used.

The hearing test was made in the following manner:

1. Air conduction levels were obtained by starting with 1000 cps. and going upward to 8000 cps., then repeating 1000 cps. and going downward to 125 cps. Masking was used only if the difference between ears was 50 db or more. The better ear was tested first.

2. Thresholds of discomfort were established for each ear, to determine the presence of recruitment in cases of bilateral deafness when there was little or no difference between air conduction levels.

3. Bone conduction audiograms were done with masking. Masking levels varied between 60-80 db. The threshold was considered the relative bone conduction.

4. Leaving the bone conduction receiver in exactly the same position, the tested ear was blocked with a piece of wet cotton, and the bone conduction audiogram was repeated. This threshold was considered the absolute bone conduction. The sum of the differences between relative and absolute bone conduction thresholds at the frequencies 250, 500, 1000 and 2000 cps. gave the "Occlusion index" (Sullivan).[8] The index ranged from 0 db, when the test was negative, to 60 db when it was strongly positive, with a mean value of 34 db for normal ears. When the difference in hearing level between the two ears was 25 db or more, a binaural loudness balance test was done to determine whether recruitment was present. The audiogram was then evaluated, and a final diagnosis was made.

FINDINGS OF THE INVESTIGATION.

1. The Bing test in normal ears:

Twenty persons were examined with normal hearing. They were 19-20 year-old student nurses of Bellevue Hospital. They had no histories of ear disease. The Bing test was positive in all these cases. The mean value of bone conduction shift for 250 cps. was 14 db, for 500 cps. 12 db, for 1000 cps. 6 db and for 2000 cps. 2 db (see Fig. 1). Adding these differences for the four frequencies, the total is 34 db (14+12+6+2).

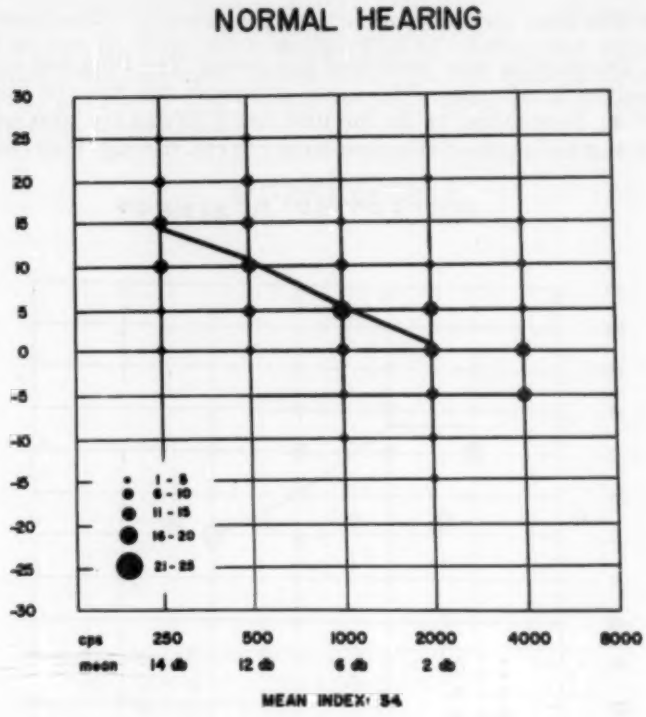

Fig. 1.

The average occlusion index for this group of normal ears is, therefore, 34.

2. The Bing test in Presbycusis:

Sixteen ears were diagnosed in this category by history, clinical examination and audiometric findings. The Bing test was positive in all cases. The mean difference for 250 cps. was 14 db, for 500 cps. 13 db, for 1000 cps. 7 db and for 2000 cps. 0 db. Adding up the differences from 250 cps. through 2000 cps., the total is 34 (14+13+7+0). The average occlusion index for these presbycusis ears is 34.

3. The Bing test in Acoustic trauma:

Twenty-four ears comprised this group. The Bing test was positive in all cases. The mean difference for 250 cps. was 17 db, for 500 cps. 12 db, for 1000 cps. 7 db and for 2000 cps. –8 db. Adding the differences from 250 cps. through 1000 cps.,

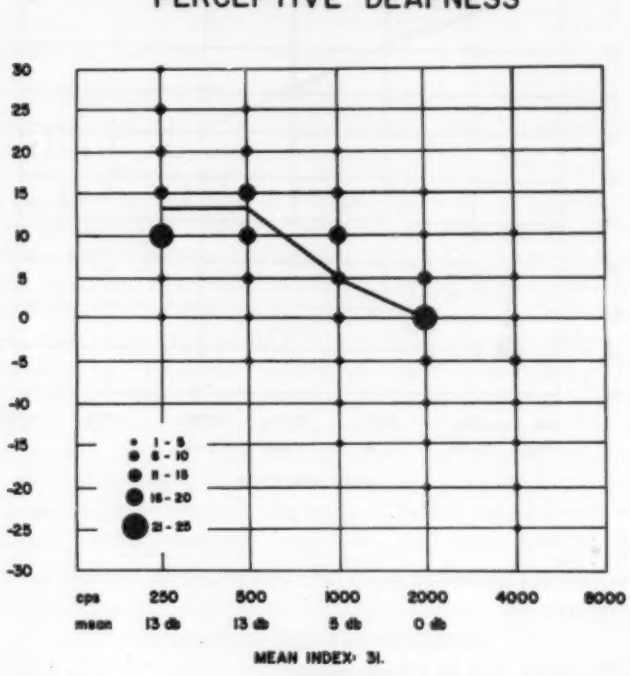

Fig. 2.

not counting the minus 8, the total is 36 db (17+12+7). The average occlusion index for Acoustic trauma is, therefore, 36.

4. The Bing test in Perceptive deafness (of different etiology):

Sixty ears were examined in this category of deafness. The Bing test was positive in all of these cases. The mean differ-

ence for 250 cps. was 13 db, for 500 cps. 13 db, for 1000 cps. 5 db and for 2000 cps. 0 db (see Fig. 2). Adding the differences from 250 cps. through 2000 cps. the total is 31 db (13+13+5+0). The average occlusion index for Perceptive deafness is, therefore, 31.

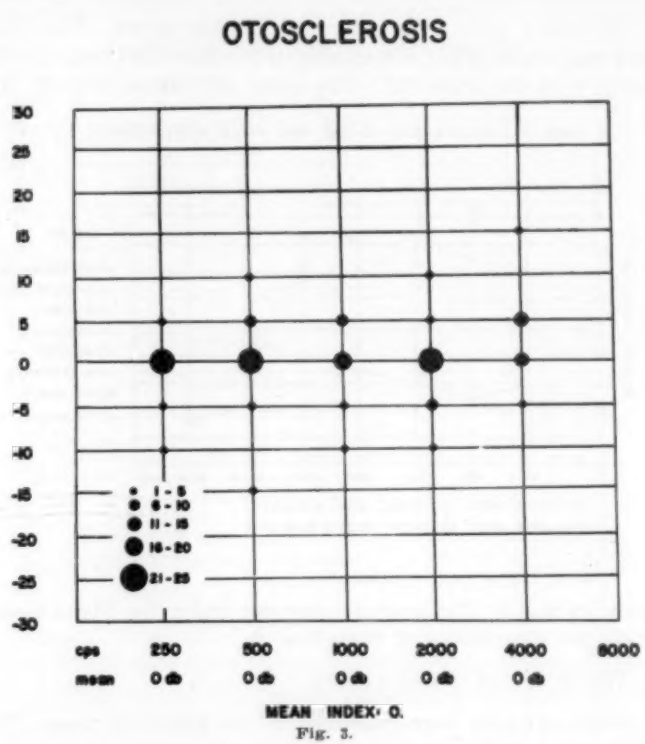

Fig. 3.

The following groups of cases have negative Bing test (no bone conduction shift):

5. *The Bing test in Adhesive otitis* (Otitis media catarrhalis chronica):

Sixty-seven ears were examined in this category. The Bing test was negative in the majority of cases and only slightly

positive in the minority. The mean value for all frequencies was 0; thus the average occlusion index for Chronic Adhesive Otitis was 0.

6. The Bing test in cases of middle ear infections (Otitis media purulenta chronica):

Thirty-six ears were examined in this group. The Bing test was negative in the majority of the cases and only slightly positive in the minority. The mean difference for all fre-

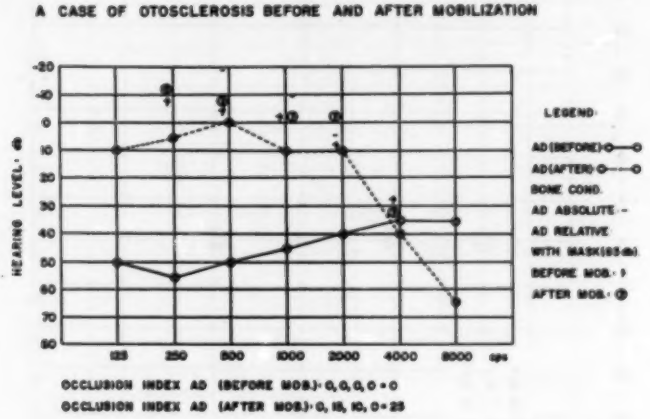

Fig. 4.

quencies was 0. The average occlusion index for Otitis media purulenta chronica was, therefore, 0.

7. The Bing test in Otosclerosis:

Thirty-six ears were examined in this group of cases. The Bing test was negative in almost all the cases. Only a few cases showed a slightly positive test with the occlusion index never exceeding 10. The average occlusion index was 0 (see Figs. 3 and 4). It is interesting to note the change in occlusion index following mobilization of stapes. Figure 4 shows a case of otosclerosis before and after mobilization. As a result of the operation the occlusion index changed from 0 to 25.

8. The Bing test in Ménière's Disease:

Twenty-eight ears were examined in this group. History, clinical and audiometric findings with auditory recruitment indicated the presence of labyrinthine hydrops. The Bing test was negative in the majority of the cases, and only in a few

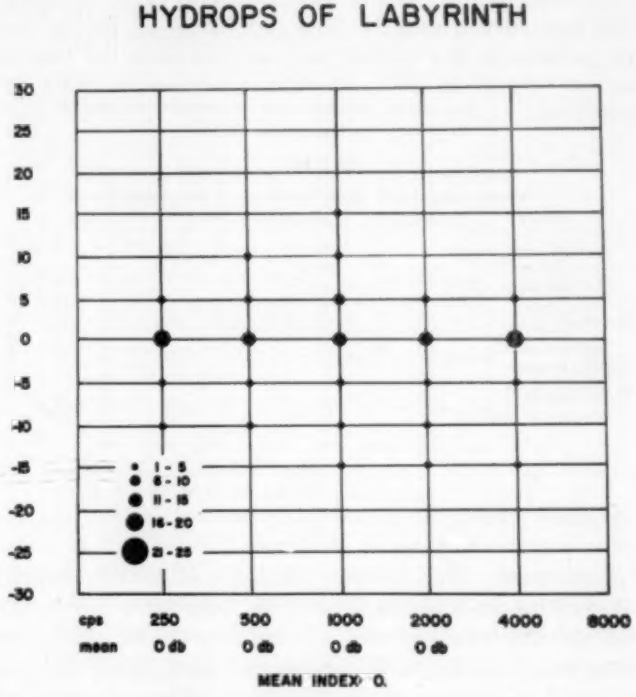

Fig. 5.

cases did we find a slightly positive Bing test. The occlusion index for all frequencies was 0 (see Figs. 5 and 6). Figure 6 shows that in the period of remission following treatment of hydrops of the labyrinth the Bing test becomes positive 35.

The average occlusion index for Ménière's Disease is 0.

Table I shows the Bing test of normal ears and of ears with different types of deafness.

DISCUSSION.

The results of this investigation show that the Bing test is equally negative in otosclerosis and in middle ear infections if we consider the average case, but mild cases of adhesive otitis may show a positive Bing test; however, not as markedly positive as in a normal ear. In such cases the Bing test may be helpful in differentiating an adhesive otitis from otosclerosis.

TABLE I.

Bone Conduction Shift in Normal Ears and in Different Types of Deafness.

Ears examined:				250	500	1000	2000		Occlusion Index:
1. Normal ears:	AD: 20	AS: 20		14	12	6	2	=	34
2. Presbycusis:	8	8		14	13	7	0	=	34
3. Perceptive d.:	30	30		13	13	5	0	=	31
4. Acoustic tr.:	12	12		17	12	7	–	=	36
5. OMCC:	36	31		0	0	0	0	=	0
6. OMPC:	16	20		0	0	0	0	=	0
7. Otosclerosis:	20	16		0	0	0	0	=	0
8. Hydrops of lab.:	12	15		0	0	0	0	=	0
	154	+ 152	= 306						

Another finding of considerable interest is that the Bing test is negative in cases of Ménière's Disease as it is in cases of otosclerosis. This indicates that the labyrinth should be considered a part of the conductive apparatus in the mechanism of hearing.

The Bing test is also helpful in the audiometric diagnosis of those less frequent cases of Ménière's Disease where both ears are involved, but one ear is more active than the other. The more active ear has a negative Bing test, whereas the less active ear has a slightly positive Bing test.

According to Aubry and Giraud[5] the relative bone conduction is from 9-12 db less acute than the absolute bone conduction in the frequencies below 2000 cps. for normal hearing persons. In early nerve deafness the absolute bone conduction

is still more sensitive than relative bone conduction, but not so much as in normal hearing persons. It is about 10 db.[8]

According to our investigation the occlusion index is the same in normal hearing persons and in persons with nerve deafness. When there is difference between these two groups, it is the author's impression that it is due to an additional middle ear lesion, usually OMCC, present in those persons with nerve deafness.

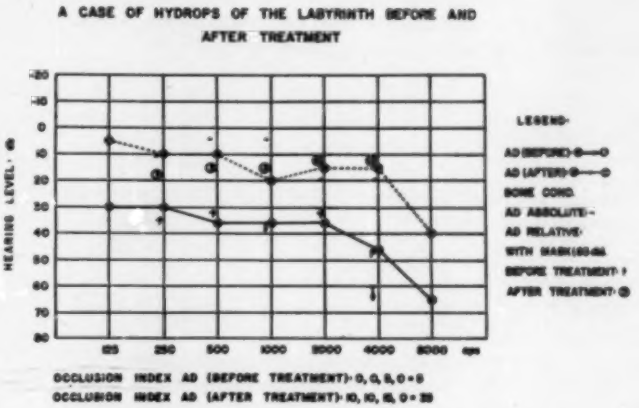

Fig. 6.

Aubry and Giraud further state that in OMPC there is little or no difference between absolute and relative bone conduction. In otosclerosis the absolute and relative bone conduction very nearly coincide. They believe that the difference in the lower frequency range between absolute and relative bone conduction is an important indication of normal middle ear function.

According to the present study this is true, but it really is an indication of any conduction lesion involving even the labyrinth as shown in cases of Ménière's Disease.

Sullivan[9] found an occlusion index of 60 in normal ears. In his series the occlusion index for cases with conductive

deafness was 0, while the index for cases of perceptive deafness was not significantly different from that for normal ears. In mixed deafness the occlusion index was significantly lower.

This author agrees with the above findings, except that he found an occlusion index as high as 60 in his series in only a few cases.

Lierle and Reger[10] found the Bing test to be "exceedingly delicate." They reported that slight middle ear involvement in which the air conduction acuity showed but little loss, was sufficient to result in a negative occlusion test.

According to the present study, this is true; in fact, it is the first sign of a conductive lesion. This is another reason why the Bing test is an important diagnostic tool.

In the same article Lierle and Reger state: "If the patient possesses a normally functioning middle ear mechanism, that is, if his tympanic membrane and ossicular chain possess a normal motility, the bone conduction acuity will be increased approximately 15 db for frequencies below 1500 cps., remaining unchanged for higher frequencies."

According to the present writer's findings, there will be no bone conduction shift in hydrops of the labyrinth. In other words, the Bing test is the measure not only of a normal middle ear function but it is also a measure of a normally functioning inner ear relating to the conductive elements of the inner ear.

Siegenthaler and Cohen[11] conducted an investigation of the bone conduction shift. They concluded that the usual explanation of the threshold change on the basis of an attenuation of environmental noise is inadequate. It is best explained by the theory of bone conduction as developed by Barany,[3,4] Békésy,[6] and Langenbeck.[7]

Markle[12] raises the question whether recruitment is an indication of a potentially reversible conductive lesion within the inner ear. He continues saying that in hydrops of the labyrinth the reduced bone conduction threshold of hearing

must be at least in part due to increased intralabyrinthine pressure. This in effect produces an increased impedence or conductive lesion within the inner ear.

The present author agrees with this statement wholeheartedly, since the Bing test, when done on patients with Ménière's Disease, indicates the presence of a conductive lesion. As soon as the intralabyrinthine pressure subsides, the Bing test becomes positive. This is an objective sign of improvement of Ménière's Disease.

Finally I close with Albert Bing's words: "I hope that my colleagues will try this test and will find it helpful among other tests in the differential diagnosis of hearing."

SUMMARY.

More than 300 ears were examined in the past four years in an attempt to evaluate the Bing test and its role in the differential diagnosis of deafness. The results of this investigation are as follows:

1. The Bing test is positive in normal ears, in presbycusis, in acoustic trauma and in other cases of perceptive deafness, in other words, when the conductive mechanism of the middle and inner ear is intact. The occlusion index in these cases was from 31-36.

2. The Bing test is negative when the conductive mechanism of the middle and inner ear is disturbed as in otosclerosis, hydrops of labyrinth, and in the majority of the middle ear lesions as OMCC and OMPC. The occlusion index is minimal (10-25) in mild cases of middle ear pathology, about 30 per cent of cases with OMCC and OMPC.

3. The Bing test is a sensitive test, which indicates a disturbance of the conductive elements of the ear even when there is no significant loss noted in the air conduction threshold.

4. The Bing test is a simple test, easy to carry out, takes little time to administer, and it is helpful in the differential diagnosis of deafness.

5. It is felt by this author that this test should be done routinely in order to arrive at a better diagnosis of hearing impairment.

ACKNOWLEDGEMENT.

I extend my sincere gratitude to Dr. Francis Kwok for instigating this work, to Dr. John F. Daly for his support and guidance in this work, to Joseph E. Hawkins, Ph.D., for his assistance in reviewing it and to Mr. Werner Blankenburg for the preparation of the drawings.

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LARYNGOCELE.*

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Baltimore, Md.

Cystic dilatation of the laryngeal ventricle in man is not a common condition, and the number of reported cases in the literature is approximately 100, up to the present date. It is of interest that almost 50 per cent of these cases were reported during the last ten years. The laryngeal ventricle was mentioned first by Galen,[1] 300 A.D.; however, it is believed that the first case of laryngocele was reported in 1829 by Larrey,[2] a French Army Surgeon.

The term laryngocele refers to the cystic dilatation of the appendix or the saccus of the ventricle, the latter being a constant anatomical finding in the human. The appendix of the ventricle varies in size, but usually it does not extend higher than the upper level of the thyroid cartilage. It is well developed in fetal life and infancy, but later lags behind the ventricle in growth.

Since laryngeal air sacs are normal anatomical findings in many animals, the presence of a dilated laryngeal ventricle in man is regarded by several authors as an atavistic phenomenon, illustrating developmental changes of evolutionary degeneration.

Negus[3] gives a detailed description of the various types which are found in different animals. He felt that the purpose of these large air sacs, like the buccal sacs in frogs, the large external laryngeal sacs in apes, or the abdominal sacs in birds, is to serve as a rebreathing apparatus, enabling the animal to hold its breath longer. Watkins[4] suggested that the laryngeal ventricle may represent a remnant of the large extra-laryngeal sacs of some anthropoid apes.

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As far as etiology is concerned, numerous factors are mentioned in the literature. They have in common the fact that the intraglottic pressure is abnormally increased in these conditions, like coughing, singing, glass blowing, horn blowing, weight lifting, etc. It is also believed that certain diseases, like tuberculosis, syphilis or tumors, may weaken the wall of the larynx and predispose it to the development of laryngoceles, especially in the presence of other etiologic

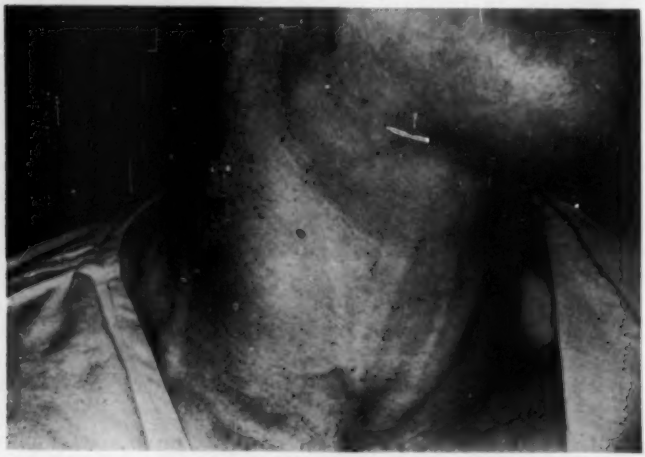

Fig. 1. Swelling of neck extending up above the level of the hyoid bone on the right side.

factors;[5] however, pre-existing pathologic condition is not necessary to the development of a laryngocele, for the condition is observed in the normal larynx.[7]

Another view is that a valve-like closure occurs in the sacculus so that air is allowed in, but not out. This can result in dilatation of the sacculus and finally can lead to the formation of a laryngocele. The congenital origin of certain laryngoceles seems to be supported by case reports describing this condition in young children of from nine weeks to 13 years of age.[6,7,8]

There are three types of laryngoceles; the most common

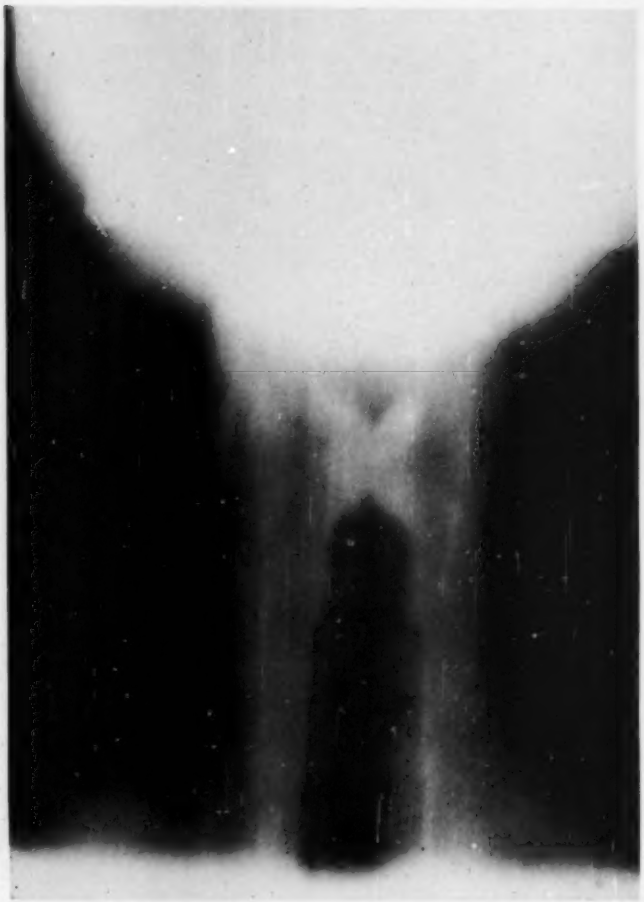

Fig. 2. Antero-posterior laminagrams of the neck demonstrate a large air-containing mass on the right side and a small one on the left side.

being the internal. It is a cystic dilatation in which the hernial protrusion of the mucous membrane lies within the larynx. The external type occurs less frequently. It appears as a swelling in the neck. The sac perforates the thyrohyoid membrane, frequently at the point where the superior laryn-

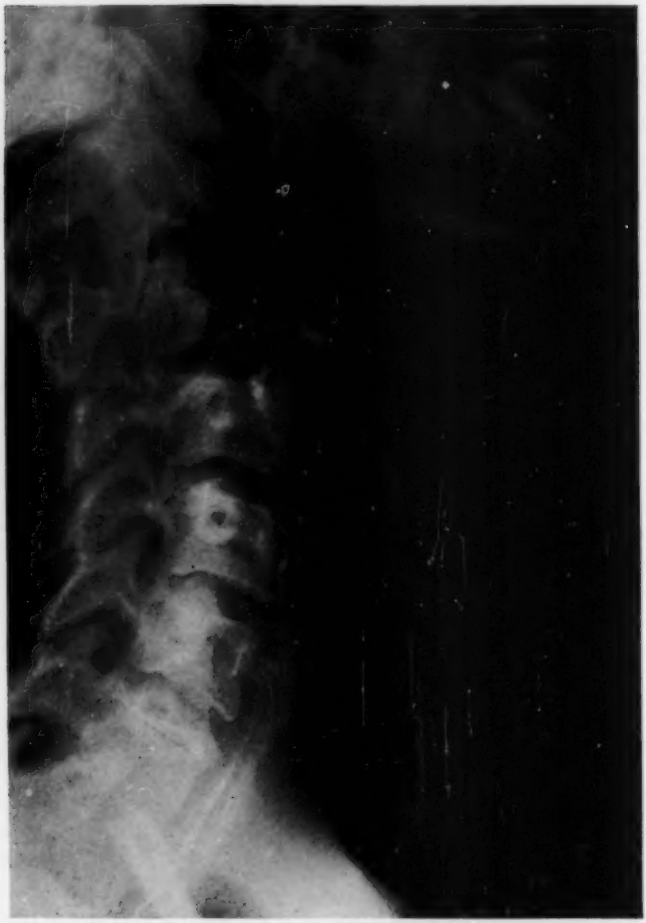

Fig. 3. Lateral soft-tissue roentgenogram of the neck shows a large air-containing mass extending up above the level of the hyoid.

geal vessels and nerve enter the larynx and projects as a hernia through the thyrohyoid membrane; however, the sac may protrude through the cricothyroid membrane, or very rarely, following an injury, or a pathological process resulting in a pathway, it may be in any area of the larynx. In

the mixed type of laryngocele the extra-laryngeal portion is frequently large and joined by a small isthmus with the internal.

Laryngoceles are lined with ciliated respiratory epithelium, and the stroma contains lymphoid cells, mixed mucous and serous tubular glands; however, in recent traumatic cases as reported by Simpson[9] and others, there was not a definite sac lined with respiratory epithelium.

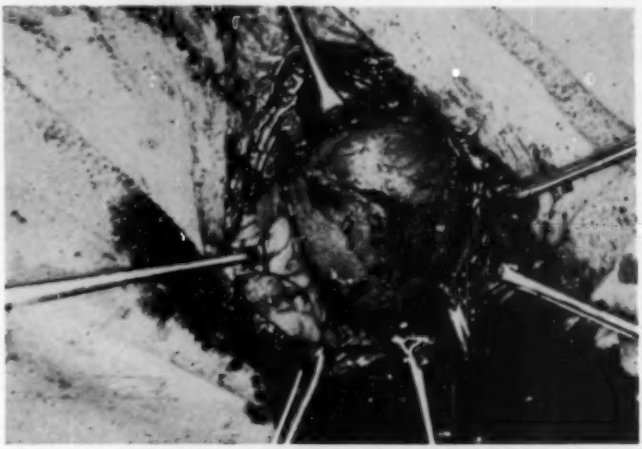

Fig. 4. The appearance of the sac after the platysma fibers were divided.

It is interesting to notice at this point that even though laryngoceles which arise in pathological larynges and from trauma and which do not have definite walls, are not followed by diffuse emphysema. There are authors who do not accept these traumatic laryngoceles as true laryngoceles where the air is caught between the tissues and held by a ball-valve arrangement and the air sac is not lined by mucous membrane, but rather by the tissue in which it forms.

It is interesting to notice that the incidence of carcinoma of the larynx in association with laryngoceles is relatively high. Leborgne,[10] who studied a large number of laryngeal

carcinomas from the radiological standpoint, feels that the incidence of associated laryngocele and cancer of the larynx may be as high as 10 or 15 per cent.

Symptoms depend to some extent upon the type of laryngocele. Laryngoceles may also be completely asymptomatic. Internal laryngoceles may dilate on forced expiration and if large, may partially or completely produce respiratory obstruction, which will necessitate emergency tracheotomy.

Fig. 5. The sac protrudes through the thyrohyoid membrane just above where the superior laryngeal vessels and nerve enter the larynx.

It can produce voice changes or hoarseness. The superior external laryngocele appears as a swelling on the neck. The swelling enlarges on coughing, and it can be emptied by pressure. Headache and local discomfort may be caused by these laryngoceles because of the compression on the large neck vessels. Accumulation of fluid in the sac is likely with periodic secondary infection with resultant coughing and expectoration of pus when the sac is compressed. The voice is not usually affected unless there is an accompanying internal laryngocele. The combined internal and external laryngocele may result in a combination of the above-mentioned symptoms.

The presence of a soft elastic mass on the neck, changing in size when the intraglottic pressure is increased and which can be compressed, and is associated with some of the symptoms previously discussed, directs the attention to this particular disorder. Direct laryngoscopic examination and roentgenological studies, especially laminagrams, are of great help in making the diagnosis.

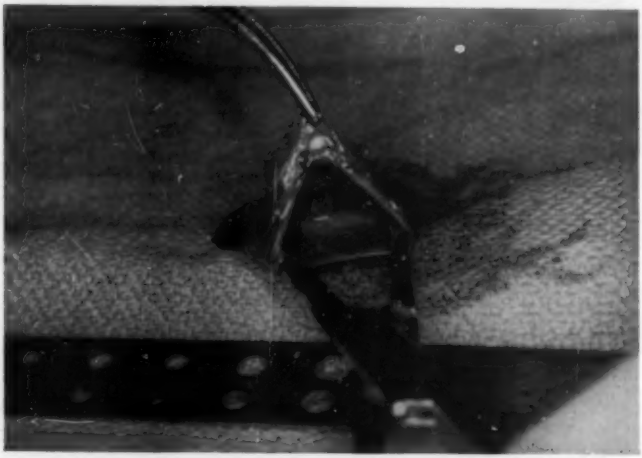

Fig. 6. The opened sac after it was removed.

Laryngoceles producing symptoms should be removed surgically. The external approach is favored for the removal of both the external and internal variety; however, small internal laryngoceles have been successfully removed through the laryngoscope. The use of cautery and injection of trichloracetic acid into the cyst also has been attempted, but should be discouraged. X-ray therapy results in failure. For marked obstruction caused by laryngoceles, intubation and tracheotomy may be required. If the laryngocele becomes infected, surgical drainage and antibiotic therapy should be instituted. When co-existing lesions are present, like papilloma,[11] granuloma or carcinoma,[12] they should be treated adequately with laryngocele.

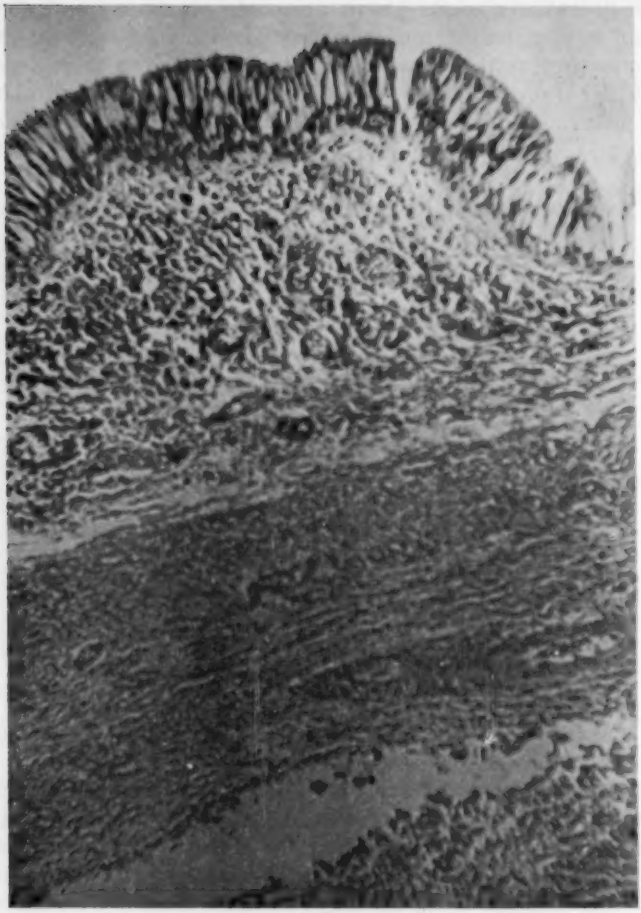

Fig. 7. Microphotograph (100 x) of a portion of the wall of the laryngocele shows pseudostratified ciliated columnar epithelium resting on a thin basement membrane. The tunica propria is made up of moderately vascular fibrous connective tissue.

CASE REPORT.

A 22-year-old white male was admitted to the Otolaryngology Service at University Hospital, Baltimore, Md., because of swelling in right neck. The patient was in good health eight months prior to admission, when following an episode of a bad cold he noticed a small swelling of the right side of his neck under the jaw. This swelling gradually increased in size, it became considerably larger on coughing, with occasional mild respiratory difficulties and changes in his voice. On pressure over the mass, the patient observed a quick reduction in its size, which was accompanied by a hissing sound.

Physical examination revealed on the right side of the neck the presence of a tumor-like swelling which readily increased in size on the Valsalva's maneuver and decreased on direct pressure over the mass, extending above the hyoid bone. Indirect and direct laryngoscopic examination did not disclose considerable anatomical changes in the larynx. Roentgen studies of the neck, including a barium swallow and frontal tomography of the neck, revealed a 6 cm. sized air-filled mass extending above the thyroid cartilage and bulging into the right pyriform sinus. There was a smaller similar lesion on the left side just above the thyroid cartilage. These air-filled structures appeared to rise from the laryngeal ventricles, and the diagnosis of bilateral external superior laryngocele was confirmed by the radiologist. On performing the swallowing function, barium did not enter the right pyriform sinus but passed down the left pyriform sinus into the esophagus.

On February 23, 1961, the large laryngocele on the right side was removed without rupture, but the small sac which was detected only on laminagrams on forced expiration against the closed nose and mouth, could not be found in its collapsed state. Under general anesthesia a transverse incision was made between the hyoid bone and the thyroid cartilage. When the platysma fibers were divided and the superficial fascia opened, a thin-walled sac appeared between the midline and the sternomastoid muscle laterally on the right. The sac was in intimate contact with the superior laryngeal nerve and vessels inferiorly, the submaxillary and lingual vessels superiorly, and the carotid sheath posteriorly. The sac was dissected down free to the pharynx where its neck could be seen coming through the thyrohyoid membrane, somewhat medially and superiorly from the point where the superior laryngeal nerve and vessels enter the larynx. The neck was then ligated, divided, and the stalk inverted with a purse-string suture. A small rubber drain was inserted and the wound closed. The postoperative course was uneventful, and the patient was discharged on the seventh postoperative day.

The pathologic report: Cyst lined with columnar epithelium. Consistent with laryngocele.

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SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY.

The Sixth International Congress of Audiology will be held in Leyden, The Netherlands, September 5-8, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary: Dr. A. Spoor.

The program will include three round-table talks on "Frequency analysis of the normal and pathological ear." Moderator: Prof. Dr. G. von Békésy. "Central deafness in children." Moderator: Prof. Dr. J. M. Tato. "Psychogenic deafness and simulation." Moderator: Prof. Dr. H. A. E. van Dishoeck, and associated and independent papers.

Official languages of the Congress are: English, French, German and Spanish. Working languages will be: English and French.

For further information address the secretariat, Ear-Nose-Throat Department, Academisch Ziekenhuis, Leyden (The Netherlands).

PROCEEDINGS OF THE EIGHTY-SECOND ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION AT THE LAKE PLACID CLUB

ESSEX COUNTY, NEW YORK

MAY 21 - 22, 1961

EDWIN N. BROYLES, M.D., President

Abstracted by

SAMUEL SALINGER, M.D.

MAN MADE AIR—PROBLEMS AND LIMITATIONS.

CAPT. GERALD J. DUFFNER, MC, USN (By Invitation).

Since the crew of an atomic powered submarine must remain submerged for long periods of time, the problem of supplying respirable air is a complicated one. It embraces not only the maintenance of an adequate supply of oxygen but also the problem of eliminating the carbon dioxide and certain toxic substances such as carbon monoxide, aerosols, heat and water vapor, hydrocarbons and toxic volatile matters such as methyl alcohol, etc. Then toxic substances originate either in the materials of which the submarine is constructed, or introduced by the crew or induced by their activities and materials employed in efforts to remove the first two. The by-products of tobacco smoke, emanation from the ship paint and cleansing materials are among a

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few mentioned. The mechanics involved in the control of these elements are all given in some detail.

MAN, DRUGS AND SPACE FLIGHT.

LT. COL. CHARLES A. BERRY, USAF, MC (By Invitation).

Dr. Berry discussed the problems affecting the reaction of astronauts in space flight. These include the oxygen, carbon dioxide and water vapor exchanges, the reaction to changes in barometric pressures, exposure to radiation, ultra violet rays, meteors, etc. Measures to adjust the flier to these varying situations were described.

The possible value of drugs in connection with flight has been considered but no conclusions arrived at insofar as their practicability is concerned. Experiments on animals cannot be interpreted as equivalent to reactions in the human. More exact information is required as to the specific mechanism of their action, their toxicity and effects on performance under the conditions of flight before they can be deemed safe and profitable. For the time being it is better that the astronaut accomplish his mission without the aid of drugs.

A HISTOLOGICAL METHOD FOR THE STUDY OF THE SPREAD OF CARCINOMA WITHIN THE LARYNX.

GABRIEL F. TUCKER, JR., M.D. (By Invitation).

Classification of malignant laryngeal tumors has heretofore depended upon the laryngoscopic visualization and roentgenologic interpretation and is usually determined by examination of the split larynx. A more accurate appraisal of the precise location of the lesion and its spread into submucosal areas can be obtained by the study of serial sections as proposed by the author. The entire larynx is imbedded in nitrocellu-

lose and serially sectioned in the coronal plane. This yields an average of from 1,100 to 1,700 sections, every tenth one being stained and mounted. The remainder may be stored for further study and special stains.

This procedure affords the opportunity of determining the relationship of the primary lesion to the underlying connective tissue, especially the conus elasticus and the laryngeal compartments.

DISCUSSION.

Dr. Joseph P. Atkins called attention to two significant aspects of Dr. Tucker's presentation. The sections in the coronal plane make his study correspond well with clinical observations by mirror and direct laryngoscopy all in the same plane. Second, these studies demonstrate the direction of the spread of carcinoma within the larynx, thus corroborating clinically the compartments of the larynx shown experimentally by Pressman. These studies may suggest a new approach for the removal of a supraglottic growth without crippling the phonatory function. He asked Dr. Tucker whether he felt that the conus elasticus might serve as a barrier to tumor spread and whether other aspects of laryngotomy might not be altered through this newer method of study.

Dr. Edwin N. Broyles called attention to the difficulty he has encountered in finding a tumor that started in the laryngeal appendix or saccule, since biopsies in this area so frequently fail to go deep enough to reach the lesion. He was impressed by the fact that Dr. Tucker presented one case of this type showing the lesion in the saccule, even though it took three biopsies to confirm the diagnosis.

Dr. Anderson C. Hilding asked whether the essayist or anyone else could answer the question as to where a squamous cell carcinoma is most likely to start: the anterior third, the middle or the posterior third.

Dr. James McNally, in complimenting Dr. Tucker, felt that mention should be made of the fact that he was a worthy son of his illustrious father whom we all knew.

Dr. Tucker closed the discussion by answering some of the questions posed. Regarding the conus, he could say only that the sections were suggestive of Dr. Atkins' observation.

He showed two more slides, both of benign tumors, one of which required a laryngectomy and the other an agioma, whose exact extension could not be accurately determined until after the tracheotomy had been performed. This case demonstrates the predilection of the cricoid area for hemangioma, especially in children.

Concerning the favorite site for onset of squamous cell carcinoma, he believes that most cases start in the anterior half, probably the junction of the anterior and middle thirds.

THE TREATMENT OF HEAD AND NECK CANCER WITH THE CONTINUOUS ARTERIAL INFUSION OF METHOTREXATE AND THE INTERMITTENT INTRAMUSCULAR ADMINISTRATION OF CITROVARUM FACTOR.

ROBERT D. SULLIVAN, M.D.,

and

JOHN F. DALY, M.D. (By Invitation).

Heretofore the systemic administration of cancer chemotherapeutic compounds in patients with incurable head and neck cancer has resulted in little benefit in the practical management of these disorders. A method of therapy is described which embodies the regional (arterial) 24-hour administration of supralethal doses of an antimetabolite (Methotrexate), together with the intermittent use of the specific metabolite (antidote—Citrovarum Factor), to prevent serious systemic toxicity. The technique of catheter insertion and fixation, maintenance of infusion assembly and dose schedules are reported.

Patients with incurable cancer and no known metastases are the most favorable for this treatment; however, when resectable nodes are present neck resection may be combined with the chemotherapy. If the metastases are widely diffused outside the distribution of the arteries catheterized (external carotids), these cases are not suitable for the therapy.

Thirty-nine patients were selected for therapy and received at least one course of therapy. Twenty-six out of 35 that were evaluated had partial tumor regression. In eight patients, whose tumor was present only in the area of therapy, there was complete tumor regression up to 15 months of observation. In several cases more than one course of therapy was given with no ill effects.

DISCUSSION.

Dr. John J. Conley explained that infusion consists of supplying a calculated and regulated supply of a drug, which is essentially a poison, in a selected artery for a given period of time, with no attempt to recover the material or prevent its entrance into the general circulation, but to combat its toxicity by an antidote which is possible with methotrexate. Perfusion involves the recovery of the drug through regional veins which is impossible when treating tumors of the head and neck, since systemic exclusion is far from adequate.

The technique is difficult, and complications from the insertion of the catheter as well as from systemic reactions and bone marrow depression run well over 42 per cent; nevertheless it is significant that 74 per cent of Dr. Daly's cases had some clinical regression, and 23 per cent showed total objective regression.

While these procedures cannot at the present time supplant surgery or irradiation, they, nevertheless, have proven of value in diminishing pain and should be encouraged even though at present they are more or less of a pilot nature. Further studies may succeed in developing a chemical that will accomplish a reduction in the growth of the tumor without the risks and dangers attending our present day procedures along these lines. Dr. Francis Lederer recalled Dr. Joseph Beck's research into what were termed "protoplasmic poisons," and the limited success attending their use. This type of investigation is not one that can readily be employed in every hospital. Dr. Sullivan, who coauthored the paper, works for the Veteran's Administration, with plenty of funds available for this type of research and is, therefore, in a position to conduct an investigation of this type under the best auspices.

Dr. Harry P. Schenck inquired whether the authors had seen any disturbances of speech due to the action of the drug on the tongue. He mentioned also that he had known of a case where there was considerable damage to the retina.

Dr. J. A. Zanka asked whether it would not be a little less hazardous and technically easier to ligate the superior thyroid artery.

Dr. Daly closed the discussion. First he cleared up a misunderstanding about the survival time of his cases. The longest period was 28 months. The longest period without any apparent disease was 15 months. Referring to hypopharyngeal lesions they tried to preserve the superior thyroid artery by putting the catheter into the bulb and fixing it to the arterial wall so the infusate would circulate through the superior thyroid artery. They have used the thyroid artery at times, but that eliminates it from the circulation. Other techniques, such as putting the catheter into the occipital artery, the external carotid, the subclavian, etc., were also tried. Dr. Daly mentioned the fact that Dr. Sullivan, in addition to his duties at the Veterans Hospital, also works at the Sloane-Kettering Institute.

He agreed with Dr. Conley that the procedure is hazardous and should be undertaken with the utmost caution and with full realization of the perils. Replying to Dr. Schenck, Dr. Daly, while admitting that the alkalating agents are very damaging, stated that they had failed to see any case of limitation of motion of the tongue; on the other hand, they had one case of a tongue lesion that was greatly benefited by the treatment.

THE PROBLEM OF STOMA CONSTRUCTION IN LARYNGECTOMY CASES.

ARTHUR J. CRACOVANER, M.D.

The difficulty of constructing a tracheal stoma has been due to a number of factors such as fistula formation, additional scarring from the neck dissection, staphylococcic infection, narrow trachea with delicate ring cartilages, displaced large thyroid lobes and prominent attachment of the sternomastoid muscles.

Suggested measures for improving the results, using the lower half of the cricoid where possible, are wide excision of the skin especially to the unoperated side, submucous excision of the anterior portion of the first two tracheal rings, developing as large a mucosal flap as possible and using silk instead of catgut sutures. The author also dispenses with the feeding tube, which causes too much irritation, provokes swallowing and tends to develop a fistula. Instead he feeds the patient intravenously for five to seven days, and then gradually permits fluids and later, solids, to be ingested.

DISCUSSION.

Dr. Julius McCall declared that he would be reluctant to give up the feeding tube, although he admitted that its prolonged use favors the development of a fistula. When a neck dissection has to be done along with the laryngectomy, the danger of a resulting fistula is enhanced. For this reason, Dr. McCall proposes placing the incision lateral to the midline and showed a short movie displaying the technique. He favors the use of the tracheotomy tube at intervals and especially at night.

Dr. Herbert Harris agreed with the author on the three points he outlined; namely, cutting back the cartilage so it doesn't lie in the junction of the skin and mucosa; the cutting of the trachea at an angle, and the taking of more skin that would appear to be required. Dr. Harris never uses a laryngectomy tube but prefers a tracheotomy tube with a cone shaped dressing.

Dr. Lyman Richards asked whether Dr. Cracovaner makes a circular incision in the skin first, as shown in Dr. McCall's movie. He does not favor the removal of the first tracheal ring since he relies on it for supporting the mucocutaneous collar.

Dr. William Montgomery stated that at the Eye and Ear infirmary in Boston they have been using Dr. Schall's U flap, dissecting away the subcutaneous fat and suturing the thyroid lobes laterally under the sternomastoid muscle. Recently they have eliminated the feeding tube,

relying on intravenous fluids for three days and then starting with sterile liquids the fourth day. They have also been using the Hemovac which is a portable suction apparatus that is put in through the drainage sites and keeps the spaces below the flap free from secretion. He feels that stenosis of the stoma is due to irritation of the tube. Using the Hemovac for three days, they have been able to remove the Hayes-Martin tube within five days.

Dr. Daniel Baker has not been so fortunate as some of the speakers, in being able to retain as much of the tracheal rings as he would like. When doing a combined neck dissection and laryngectomy he removes the isthmus of the thyroid gland and a portion of the lobe on the operated side. It has been his experience that palpable nodes in the thyroid are one of the causes of a narrowing stoma. Contrary to the opinions expressed, Dr. Baker prefers that his patients wear the laryngectomy tube as large as possible and for as long a period as possible. In order to create a large stoma he transects the uppermost tracheal ring to spread it, while suturing the ring below to the skin of the neck, thus producing an enlarged oval stoma when the skin and mucosa are united over the split ring.

Dr. Cracovaner, in closing, thanked Dr. McCall for showing his movie. As for the feeding tube, he got the idea of leaving it out, from the Mayo Clinic, where they reported discarding the tube in cases of esophageal diverticulum. Concerning the nutrition of the patients, they do give them proteins intravenously, but do not start liquids orally until the fifth day. As for Dr. Harris' preferring the tracheotomy tube well protected, he (Dr. Cracovaner) accomplishes the same result with the laryngectomy tube, but takes extra precaution against friction by using an ace bandage around the neck. Replying to Dr. Richards' query as to the type of incision, the diagram he showed places it along the level of the hyoid bone and alongside the neck, going over the midportion of the clavicle. The ring of skin is excised when he is ready to sew the trachea to the skin, and in his experience, the mucosa and skin are not so delicate as Dr. Richards had found them.

Concerning Dr. Baker's suggestion re the thyroid gland, he also removes the gland on the operated side. In any case it is important to relieve the trachea of any outside pressure such as the gland might exert. As for splitting one or two of the rings, as suggested by Dr. Baker, it has been his experience that these split rings tend to approximate later, due to their natural spring.

SURGICAL DECOMPRESSION OF THE RECURRENT LARYNGEAL NERVE IN IDIOPATHIC UNILATERAL VOCAL CORD PARESIS.

Preliminary Report.

JOSEPH H. OGURA, M.D.

Unilateral vocal cord paresis may be termed idiopathic when all of the usual etiologic factors have been eliminated.

The author believes such cases of peripheral paresis are due to a physiological block of transmission of nerve impulses. The anterior branch of the recurrent laryngeal nerve passes into the larynx just anterior to the crico-thyroid articulation where, under certain circumstances, it may be compressed. Subperichondrial resection of thyroid cartilage at this point may result in decompression of the nerve and restoration of function.

The technique is described and four cases cited, in two of which a dramatic return of function was achieved. Both of these were operated about four months after the onset of the paresis. The two unsuccessful cases were done at later periods, which suggests that in these idiopathic cases one should not wait more than four months before resorting to surgery.

DISCUSSION.

Dr. Louis H. Clerf has found, after a study of a large number of cases due to various causes, that if recovery is to ensue, it usually manifests itself by the end of the third month, and that persisting paresis, up to six months, means that the status will remain unchanged. Dr. Clerf is not satisfied with our terminology. "Cadaveric" means just that, namely the position of the cords in the cadaver and should not be applied to the living. Paresis is either partial or complete, depending upon the extent of the interruption of the nerve pathways. As to the space between the thyroid and cricoid, he had never visualized it as being so narrow as to constrict a nerve passing through, and would like Dr. Ogura to explain it further and to declare whether the nerve is pinched because of its proximity to the cricothyroid articulation.

One must eliminate the possibility of a coincident spontaneous recovery at the time of surgery, since in two of his cases this happened at four and five months. On the other hand, the rapid response following surgery would discount the probability of spontaneous recovery which develops at a slower rate.

Chairman Theobold recalled two cases following surgery: one on the back, and the other, an appendectomy. He wondered whether traction on the neck during the anesthesia might not have been a factor.

Dr. Theodore E. Walsh had seen these cases of Dr. Ogura's and was impressed by the immediate response in the boy's case. He thinks the situation is parallel to that of a Bell's palsy, where spontaneous recovery ensues in many cases, but where, beyond a certain period of time, hope of recovery is lost. Early decompression is done when the spontaneous response is slow, even though it is possible that it might still develop.

Dr. Joel J. Pressman had hoped that Dr. Clerf would refer to his previously well turned phrase that "Semon's law had been repealed."

This concept, together with Dr. Murtagh's work on the anatomy of the nerve, have clarified much of our thinking on the subject. The confusion in the matter of describing the position of the cords is due to a failure to consider the basic principles involved. One must differentiate a paralysis of central origin, which is spastic, from one of peripheral origin, which is flaccid. Abnormalities in the movement of the cords also may be due to hyperstimulation from irritation of the nerve, as in thyroidectomies or in crico-thyroideal spasms which are of brief duration. Dr. Ogura's observations add another facet to our knowledge of the subject of laryngeal paralysis. Chairman Theobald requested Dr. Ogura's opinion about the two cases he mentioned.

Dr. Francis L. Weille asked that Dr. Ogura clarify one point, namely whether the incision he described, which is at the level of the external branch of the superior laryngeal nerve, and the removal of the cartilage taking away the insertion of the cricothyroid muscle, has any effect on the function of this muscle. Dr. Godfrey E. Arnold commented on the variety of paralyses that may involve the larynx, due to so many causes resulting in rapid or slow development that may be partial, incomplete or total. That portion of Semon's law, referring to slow progressive lesions affecting the abductor fibers first, is still valid, although his other concepts have since been repudiated. The operation described by Dr. Ogura should be done early, since chances of recovery diminish after six months.

Dr. Cgura replied to Dr. Clerf's question by stating that anatomic variations are quite common in various areas of the body, and so the space between the thyroid and cricoid may be much narrower in some, particularly near their articulation, where the nerve can be compressed. This was found to be the case in four of his patients. There is no harm in doing the operation, even if spontaneous recovery may be possible—yet, to obtain the best results it must be done somewhere between the fourth and fifth month.

A DISCUSSION OF FORTY-TWO YEARS' EXPERIENCE WITH OTOLARYNGOLOGICAL PATHOLOGY.

ANDREW A. EGGSTON, M.D.

After commenting on the virulent infective processes so prevalent in the preantibiotic days, Dr. Eggston draws on his vast experience to discuss several interesting pathologic entities in the otolaryngological field. These include papillomatosis, glomus tumors and neuroblastoma. He pointed out the differences between polyposis and papillomatosis involving the sinuses and showed how the latter is frequently mistaken for carcinoma. Of course, the papilloma may degenerate into malignancy if neglected, as shown in one case cited.

Glomus tumors are considered as hemoreceptors as well

as chemodectors. Cases involving the middle ear and the carotid body were presented, demonstrating not only the pathologic features but also the serious implications of uncontrolled extension into areas where eradication is difficult.

Tumors of neurogenic origin present many difficulties in diagnosis and as a rule have a doubtful prognosis. The cases cited are unhappy examples.

DISCUSSION.

Dr. John F. Daly paid tribute to Dr. Eggston's brilliant career and invaluable service to the Manhattan Eye and Ear Infirmary. It is significant that he has reminded the otolaryngologist that he has passed beyond the era of infections and has expanded the old Koch postulate of disease entities having a single specific cause, to the broader view of the interaction of various factors such as heredity, immunity, virus and chemical influences in the causation of disease. Another point brought out by Dr. Eggston is the value of the pathologist's greater interest and knowledge of the clinical side of medicine, which enables him to go beyond the mere interpretation of the slide.

Dr. Baker would have Dr. Eggston include in his discussion lethal granuloma, a disease which presents a challenge to the pathologist. This report of Dr. Eggston's points to new horizons which are intriguing and are adding much to our knowledge of disease. These are the studies of immune reactions, tissue transplants and the use of electron microscopy and histochemistry.

TRENDS IN THE MANAGEMENT OF PAROTID LESIONS.

GEORGE SHIMO, M.D.,
and
HARRY S. DOLAN, M.D. (By Invitation).

The diagnosis and therapy of parotid lesions are complicated and frequently inconclusive, because of the anatomic complexities in the relationship of the gland to the facial nerve and other adjacent structures as well as to the difficulty in interpreting pathologic findings. The division of the gland into superficial and deep lobes is not so sharply defined as many would believe, because there are numerous connecting strands which enmesh the branches of the facial nerve and often render cleavage more difficult.

Mixed tumors have a tendency to recur at multiple sites. This may be due to an ill defined capsule or to small excrescences of tumor tissue which are shown to protrude through the capsule. Extensions of the growth into adjacent areas are explained by the anatomy of the region.

Malignancies still present a problem in diagnosis and therapy. A combination of radical surgery including resection of the nodes and irradiation, offers the only prospect of a cure. Unfortunately, up to date, it has proven effective in only a limited number of cases.

DISCUSSION.

Dr. Albert C. Furstenberg's comments were restricted to a consideration of pseudoadenomatous basal cell carcinoma, otherwise known as cylindroma adenomyoepithelioma, adenocystic basal cell carcinoma, etc. Over a period of years he has collected 65 cases, 14 of which involved the parotid gland. The condition is twice as common in females as males, and the parotid lesions seemed to appear in younger individuals. Chest metastases occurred in 30 per cent of the cases, often appearing as late as 12 years. Dr. Furstenberg showed some slides illustrating the pseudoacinar pattern which was noted in 81 per cent of his series. Nerve trunk sites were identified in 29 cases with perineural lymphatic extension in 28 of them.

The prognosis is generally unfavorable, only 14 cases remaining free of disease, of which only four survived beyond five years.

Dr. Furstenberg strongly urges the use of high voltage irradiation since some of thes tumors at times display some degree of sensitivity to it. Dr. Shimo agreed with Dr. Furstenberg that the cylindroma is a provocative type of growth, and that perineural lymphatic extension occurs more frequently than would appear at the first examination.

LARYNGEAL AND BRONCHIAL CANCER: A STUDY OF DOUBLE PRIMARY MALIGNANCY.

PAUL H. HOLINGER, M.D.,
KENNETH C. JOHNSON, M.D.,
DIETRICH HURZELER, M.D.,
and
ROBERT J. JENSIK, M.D.

The authors report 16 cases of multiple primary malignancy involving the larynx and bronchi. The larynx lesion

preceded the bronchial tumor in 11 of the 14 cases that were metachromous, in two cases they were synchronous, and in three the bronchogenic tumor preceded the laryngeal. The criteria suggested by Billroth, who first reported such a case, were adhered to, except the final one which insists that each tumor must produce its own metastasis. The reason this criterion is rejected is because the second primary tumor may not have metastasized. The other criteria which are valid are that each tumor must have a distinct separate histologic identity and that the tumors must arise in different locations. The cases are all listed with brief résumé of therapy applied. In the 14 cases that were metachromous the two lesions were separated by periods of from one and one-half to 15 years.

DISCUSSION.

Dr. F. Johnson Putney believes that multiple malignancies are due to a basic predisposition of the tissues, although the lesions need not necessarily appear simultaneously. In 1,442 patients with carcinoma of the larynx seen at Jefferson Hospital, 4 per cent showed two or more primary lesions. Ten out of 328 cases (3.2 per cent) had primary malignancies outside of the larynx. It is likely that more and more cases of multiple malignancies will be observed as time goes on, due to the increasing number of cases of carcinoma of the larynx which are cured by improved techniques.

Dr. Charles M. Norris found records of 15 cases of carcinoma of the lung, concurrent with or subsequently at, the Jackson Clinic during a period of 13 years. Difficulty in assessing some of these cases arises from the criterion of two different types of pathology in the two areas. This would rule out squamous cell carcinoma, which commonly occurs in both places; however in eight of their cases, death occurred from pulmonary carcinoma without any recurrence or glandular involvement in the neck. These can be considered primary independent lesions, since isolated metastases to the lung are unusual without cervical node or secondary neck involvement.

Multicentric lesions may occur in the lower respiratory tract, bearing in mind Auerbach's work on the occurrence of multiple foci of carcinoma *in situ* and actual invasive carcinoma in the tracheobronchial tree in autopsy cases of broncheogenic carcinoma.

The matter of implantation is very important, and Dr. Norris asked for Dr. Holinger's opinion in this matter. Endotracheal anesthesia may be hazardous when the tube passes through the diseased larynx. Dr. Anderson C. Hilding would like to know how many of Dr. Holinger's patients were smokers. As Dr. Putney pointed out, one must consider the entire epithelial structure, bearing in mind the continuous movement of the mucous stream and the obstructions over which it must pass or circumvent, all offering favorable sites for the development of the malignancy. The fact that most of the growths appear at the anterior portion of the cords where the mucous blanket meets a resistant obstruction, would substantiate the importance of this physiologic phenomenon.

Dr. Holinger replied to Dr. Hilding's query about smokers among his cases by stating that they had not documented this factor accurately, although they know that they were all smokers but did not know to what extent; however, Dr. Windham did mention in his report on double primaries, that these occurred mostly among heavy smokers.

He has been well aware of the danger of implantation and recently has been doing the tracheotomy first, and then introducing the anesthesia through the tracheostomy for the laryngectomy.

Dr. Putney's and Dr. Norris' observations on the histology are apropos. One cannot accurately differentiate the lesions in some cases, because basically most of them represent some portion of the epithelium. The anatomic description of the lesion is academically important. Clinically, these lung cases were solitary lesions that had to be handled as any other primary lesion, by pneumonectomy, lobectomy etc.

DEFICITS IN IRRADIATION THERAPY FOR CARCINOMA OF THE LARYNX FOLLOWED BY SURGERY.

HERBERT H. HARRIS, M.D.

A series of cases of carcinoma of the larynx treated primarily by irradiation with 200 K.V. or 2 M.E.V. in which surgery was subsequently necessary, is analyzed with respect to possible deleterious effects of the former on the operative results. The cases were divided into four groups according to the extent of the primary lesions. It was noted that in eight cases of small isolated cord lesions, subsequently operated, all but three recovered without incident. One case, in which neck resection was done, was followed by edema of the larynx necessitating a tracheotomy. The other two suffered some local necrosis. It is interesting to note that these eight cases represent a high percentage of the 20 originally irradiated for the same type of lesion.

Three other groups of cases in which the lesion was more advanced, which were irradiated and then operated upon, showed higher percentages of bad results in proportion to the extent of the original lesion. It is apparent that when irradiation is given primarily in the expectation of a cure, the risk of complications is greater should surgery be indicated at a later date, than if the lesion had been operated upon in the first place.

DISCUSSION.

Dr. John J. Conley pointed out that the incidence of complications is in direct proportion to three factors, namely the size of the part, the size of the dose and the time lag. The smaller the part, the less the risk. As to dosage, anything over 5,000 r. renders the tissues more susceptible to necrosis. As to time lag, the longer the interval between irradiation and surgery, the greater the danger of a breakdown of the tissues due to greater devitalization from the fibrosis.

An important point is the degree of accessibility to accurate appraisal of the lesion during and after the therapy. When the lesion is elsewhere than on the cord, one may be deceived by the surface appearance, since so frequently the lesion extends submucously and is not readily observed.

One might envision a plan whereby combined irradiation followed by surgery would yield a higher percentage of cures, but this would be irrational in view of the fact that in a certain number of cases, a cure has followed irradiation alone. It seems apparent that when the lesion is other than strictly cordal, the danger of complications from post irradiation surgery is enhanced. Dr. Conley would like an amplification of the statistical analysis of Dr. Harris' second group.

Dr. Joseph P. Atkins found that the statistics in the first two groups of Dr. Harris' cases were more informative than in the last two groups. His experience in 32 cases of early carcinoma which were irradiated, with only four subsequently requiring surgery, seems better than that of Dr. Harris' group of 20, in which eight cases had to be operated. This may be due to differences in the ages of the patients in the two groups. Dr. Harris' patients were nearly all under the age of 65, whereas in Dr. Atkins' cases they reserved irradiation only for patients age 65 and beyond. They prefer not to irradiate young individuals and thus avoid the risk of carrying heavily irradiated tissues for many years.

Dr. Harris agreed with Dr. Conley that tumors, other than those of the vocal cords, are unpredictable in their growth, and called attention to the fact that he had recommended irradiation only in lesions of the epiglottis and tongue. While he was disturbed over the poor results of their therapy, he found some consolation in reports of similar results by competent British radiotherapists.

Generally where possible he would prefer that surgery precede irradiation.

The matter of age, which Dr. Atkins brought up, may require further study. The fact that surgery is supposed to yield 85 per cent cures, while irradiation has yielded only 65 per cent, may suggest a re-evaluation of these methods.

FIVE YEARS' EXPERIENCE WITH THE OSTEOPLASTIC FRONTAL SINUS OPERATION.

R. L. GOODALE, M.D.,
and
W. W. MONTGOMERY, M.D.

The results of five years' experience in 31 cases of frontal sinus disease of various kinds by the osteoplastic approach,

have proven its worth, as compared to procedures previously employed. It has the advantage of affording a complete exposure of the sinus cavity which can be dealt with according to the pathology. When indicated, as for instance in the removal of a benign tumor, the mucosa and duct may be left intact, and the operation leaves no cosmetic defect. When the pathology is such as to necessitate the exenteration of the sinus contents, the cavity can be obliterated by filling it with fat, which the authors have shown remains undisturbed and is not absorbed. Several cases are cited, and the technique is fully demonstrated by a series of drawings.

DISCUSSION.

Dr. Harry P. Schenck conceded that the approach described by Dr. Goodale represents a significant advance because of its safety, cosmetic acceptibility and preservation of the orbital structures. Surgery of the frontal sinus must be considered as a separate entity. Attacks on the ethmoid and sphenoid should be undertaken by the orthodox intranasal route. Dr. Schenck still believes that every attempt should be made to preserve the nasofrontal duct whenever possible, to maintain a normally functioning sinus. This he has accomplished in many cases by the use of a 10 mm. tube which is left in place for six weeks. He then showed the procedure he follows in opening the sinus from below, dealing with the pathology and preparation of the duct for the tube.

As for the fat implant, he is not yet convinced, but still has an open mind on the subject. He agreed that one may break down the intersinus septum without fear of infecting the other sinus, as long as that sinus has a normal nasofrontal duct.

Dr. Fred W. Dixon has always had a great respect for the nasofrontal duct, and in most cases has approached the sinus by the intranasal route. Proper ventilation is obtained by removing intranasal obstructions and employing antibiotics and suction. While some of these patients may have a recurrence occasionally they are easily controlled. He would like Dr. Goodale to state his indications for the procedure he described in cases of chronic sinusitis. Dr. W. Likely Simpson advises taking care of intranasal conditions such as deviated septum or surgery on the antrum first before attacking the frontal sinus. He has done practically all of his frontal sinus cases under local anesthesia, with the aid of HMC with full cooperation of the patient; also he combines the frontal sinus operation with a complete ethmoidectomy. In extremely large frontals he uses an auxiliary incision at the upper limit of the sinus for better visualization of the interior. As far as fat implants are concerned, he has never felt the necessity of using them, since a wide exenteration with adequate intranasal drainage and the use of antibiotics has always yielded him the best results. Dr. Lyman G. Richards raised the question about concommitant disease in the ethmoid. Does the latter require a separate surgical procedure as suggested by Dr. Dixon? He would like Dr. Goodale to clarify this point.

Dr. Charles Grace reported excellent results from the use of an indwelling gold tube to maintain the patency of the nasofrontal duct,

having used them in seven cases. Only two of them have been expelled, while the rest are *in situ* and well tolerated. Dr. Samuel Salinger called attention to Dr. Ogura's report wherein he demonstrated osteogenesis in a fat implant. Four years after the operation it became necessary to open the other frontal sinus which was diseased. Since the intersinus wall was eroded, he enlarged the opening and took a biopsy from the fat implant. He showed a histologic section which clearly demonstrated osteoid tissue. Dr. Salinger had the feeling that the same would be true of Dr. Goodale's cases, if he just waited long enough. Dr. Goodale, in closing the discussion, added that Dr. Tato had told him one could probably obtain the same results using Gelfoam or blood clot. As for osteogenesis, he has as yet failed to demonstrate it, but perhaps, as Dr. Salinger said, it may in time be proven.

Dr. Goodale did not want to leave the impression that he is committed exclusively to this procedure. One has to be guided by the type of pathology encountered as well as the conformation of the sinus. As for the tubes, he has had indifferent results with them. Certainly, if a tube is to be left in for an indefinite period, one cannot say that the patient is cured, as long as he has to retain a prosthesis. He agrees with Dr. Simpson in regard to the other procedures required as a preliminary to the frontal sinus operation. For anesthesia he has been very happy with the intratracheal method which has been highly successful. He still adheres to Dr. Mosher's dictum to respect the virginity of the nasofrontal duct and avoids traumatizing it in any way. When the duct becomes stenosed from disease or trauma, it is better to obliterate the sinus than to attempt to maintain a passage into the nose by tubes. As for the ethmoid, he agrees that when involved, it should be dealt with in a separate procedure.

THE ELECTRONIC SYNCHRON-STROBOSCOPE: ITS VALUE FOR THE PRACTICING LARYNGOLOGIST.

HANS VON LEDEN, M.D. (By Invitation).

Since the human eye is capable of perceiving only about five separate pictures per second it is inconceivable that it should be able to evaluate speeds of 100 to 1,000 excursions per second, as produced by the vocal cords in action. The disc stroboscopes of former days have given way to more modern electronic devices, one of which, designed by Dr. Rolf Trincke of Hamburg, Germany, has been employed by the speaker for his daily office examinations. The apparatus consists of a microphone applied to the patient's neck just below the larynx, the light source, an electronic control unit, and a foot pedal. The fundamental frequency of the sound produced in the larynx is transmitted via electronic flashes

to a xenon lamp which emits an intermittent bead of white light at the same identical rate. This synchronization results in a still picture which accurately delineates the position of the cords during phonation. Slow motion can be produced by varying the rate of illumination in relation to the frequency of vibration, so that each successive light impulse strikes a different phase of the vibratory cycle.

The slightest variations in structure and mobility can thus be clearly visualized so that the indications for therapy may be intelligently directed.

DISCUSSION.

Dr. Albert H. Andrews, Jr., commented on the application of engineering principles to medical science, as shown in Dr. Von Leden's stroboscope. The neon tube has greatly enhanced the value of the apparatus in supplying a light of sufficient intensity. He personally has been using a disc type of laryngoscope for years and finds it very satisfactory, despite Dr. Von Leden's disparaging comment on it. With it he is able to see all the way down to the carina. The substitution of electronic sound control gives a more realistic sound to the stroboscope and makes it easier to adapt the scope to the patient's pitch and vice versa.

The automatic adjustment of the stroboscopic speed to the pitch of the patient's voice is an important attribute of this instrument.

Dr. Andrews would like to have heard more about Dr. Von Leden's clinical experience with the machine. He was impressed by the variation in pitch which could be visualized though not heard. Dr. Andrews has found the stroboscope helpful in noting vibratory movements of the cords in benign tumors and impairment of this function so often seen in malignancies, which is a very helpful diagnostic means; also following removal of cordal growths, it is interesting to follow the healing and thus to advise the patient properly as to the resumption of vocal activities.

Dr. Kneed Faaborg-Anderson agrees that the stroboscope furnishes much more information than indirect laryngoscopy, though perhaps less than high speed photography. Another approach to the study of vocal cord movement is by the Glottigraph designed by Dr. Sonderson of Lund, Sweden. Its application is still too recent to afford its proper evaluation.

He asked Dr. Von Leden how many different pitches he has investigated in routine examinations and how he changes the tone volume.

Dr. Edwin N. Broyles inquired as to whether any local anesthesia is used. Dr. Von Leden replied to Dr. Andrews that perhaps his criticism of the disc stroboscope was unwarranted, but made the excuse that he felt he was not quite as adept with his hands and eyes as Dr. Andrews.

He appreciated the comment about the value of the instrument in differentiating benign from malignant growths: also in the matter of advising vocal rest following surgery. He believes that at times this rest period is too prolonged.

Replying to Dr. Broyles, he stated that one needs no more preparation for the use of the stroboscope than for any routine mirror inspection.

Regarding the pitch employed, he has the patient elect any continuous sound, and views the larynx at this level with both moving and still pictures. The pitch is then changed and a record of it kept for future reference. This is easily done since the meter records the pitch accurately. The intensities are also varied and recorded. He has noted that vibratory disturbances may be found at certain levels and absent at others.

For those interested in stroboscopy he recommends a book by Dr. Emil Shankow of the University of Erlangen.

SARCOMA OF THE LARYNX.

CHARLES M. NORRIS, M.D.,

and

AUGUSTIN R. PEALE, M.D.

Eight cases of malignant tumors of mesenchymal origin comprising four cases of fibrosarcoma, one neurogenic sarcoma, one Hodgkin's, one reticulum cell sarcoma and two carcinosarcomas, illustrate the difficulty in making an early diagnosis. In only one of the first five cases mentioned above, did the final diagnosis agree with the initial diagnosis. The importance of obtaining a satisfactory pathologic diagnosis is stressed because it is shown that the well differentiated tumors, if small, can be satisfactorily treated by partial resection. The more anaplastic types require more radical surgery. Neck dissection is seldom necessary, since these tumors do not metastasize by way of the lymphatics.

DISCUSSION.

Dr. Daniel Baker found a record of only five cases of sarcoma of the larynx at the Presbyterian Hospital: four were fibrosarcomas and one a chondrosarcoma. The results were not good, since four of the patients succumbed. He feels this may have been due to the fact that the cases were seen too late. Inasmuch as these tumors develop in the connective tissues, the lesion may not appear on the surface of the mucosa. In such cases a submucosal mass should be explored via an external approach. He noted that Dr. Norris reported two cases of pseudosarcoma which presented a histologic appearance of squamous cell carcinoma. This is in keeping with the report of three cases presented before this society three years ago.

Dr. Baker recited the history of a case of leioma of the larynx which was very extensive and was removed via an external approach. The primary result was satisfactory.

Dr. George A. Henry reported his experience in one case in which the biopsy revealed a spindle cell sarcoma. A laryngectomy and neck dissection were done. The glands were apparently free from disease. Three years later the patient developed pneumonia at which time X-ray pictures showed two growths in the right lung. The bronchial washings produced cells similar to the original growth, and the lungs contained several metastatic growths of the same character.

Dr. Norris recalled Dr. Baker's paper of a few years ago, on the subject of pseudosarcoma, which is germane to the topic under discussion. It should caution the pathologist to examine carefully the epithelial layer, because pseudosarcoma shows more mitoses and more bizarre nuclei and even larger numbers of giant cells than the actual fibrosarcoma; so the most useful criterion of pseudosarcoma is the associated squamous cell sarcoma.

Dr. Henry's case was interesting, especially in the appearance of tumor cells in the bronchial washings, which is unusual with connective tissue tumors.

NINTH CONGRESS OF THE PAN-PACIFIC SURGICAL ASSOCIATION, OTOLARYNGOLOGICAL SECTION.

The Ninth Congress of the Pan-Pacific Surgical Association Section on Otolaryngology will be held in Honolulu, Hawaii, November 5-13, 1963.

The First Pan-Pacific Mobile Educational Lecture Seminar will be held November 13-December 10, 1963, in New Zealand, Australia, Thailand, the Philippines, Hong Kong and Japan.

All otolaryngologists are cordially invited to attend both of these meetings. The Ninth Congress offers an extensive otolaryngological program with leading international otolaryngologists participating.

The Seminar through the Pacific area offers, for the first time, scientific meetings in each country presenting medical material unique to the areas.

For further information, please write Dr. F. J. Pinkerton, Director General, Pan-Pacific Surgical Association, Suite 570, Alexander Young Building, Honolulu 13, Hawaii.

A CASE OF FIRECRACKER-INDUCED HEARING LOSS.*

W. DIXON WARD, Ph.D.,

and

ARAM GLORIG, M.D.,

Los Angeles, Calif.

There probably is not an otologist in the country who doubts that firecrackers can produce acoustic trauma. When a high-frequency tonal gap is discovered, particularly if the gap is unilateral, it is often true that the most reasonable explanation that can be extracted from the patient's history is an accidental explosion involving gunfire or fireworks. For example, in a recent survey of the hearing status of 1200 enlisted men at a naval air station,[1] nine individuals reported an incident involving a firecracker when asked, before their hearing was tested, "If there is a particular incident of any sort that you think might have damaged your hearing (for example, an accidental explosion, a blow on the ear, or the like), please describe the incident below, mentioning about how long ago it happened." We suspect that if the question had included the word "firecracker" the rate of mention would have been even higher. Only three of these nine men had normal hearing (within 20 db of audiometric zero from 1000 to 8000 cps.). In the three cases in which the subject indicated which ear was nearer the explosion, this ear was indeed the poorer ear. Such evidence is fairly compelling in inducing one to ascribe the hearing loss to the firecracker; however, such a diagnosis is by no means certain. The same type of audiogram observed in the men just mentioned can be found in persons whose loss is—again in terms of the "best guess"—due to industrial noise, scarlet fever, or even "congenital perceptive deafness."

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The problem, of course, is one of control: we know only what the hearing level *is*, not what it was before the incident. Ordinarily, persons who suffer acoustic trauma are not considerate enough to have audiograms taken just before the accident. A diagnosis is, therefore, usually based on the assumption that the hearing was "normal" just before the blast; however, in the case of a unilateral high-frequency gap that starts at 4000 cps. or higher, this assumption is often questionable, since patients with such losses are often completely unaware of them, and may have had them for years before the incident occurred. For this reason, we feel it is worth while to document even a single case of acoustic trauma for which a very reliable pre-exposure audiogram had been obtained.

The individual concerned, G. R., age 21, had for two years been a member of a group of students employed in our experiments on temporary threshold shifts from noise.[2,3] In that period of time, about 50 pre-exposure audiograms had been taken. On April 28, 1959, during the annual madness that becomes epidemic around universities in the Spring, a firecracker (an ordinary flashlight cracker about two inches long and 3/16 inch in diameter) exploded in his hand just as he was preparing to throw it at the enemy (the fraternity next door). At the time of explosion, the firecracker was apparently about 15 inches from his right ear, in line with the interaural axis.

Because he was accustomed to having a noticeable hearing loss for a day or two following exposure to our laboratory noises, he was not at first concerned either about the fact that speech sounded muffled in the right ear or about a fairly loud tinnitus in this ear; however, when after two days these symptoms showed no signs of diminishing, he sought medical aid. We did not see him until a week after the accident, since he assumed we would not want him to listen to noise with a pre-existing threshold shift. After we convinced him that we were as interested in impulsive noise as in steady noise, he came in for testing at regular intervals. Now, two years after the accident, the hearing is at least stable.

Figure 1 shows the average threshold sensitivity before the accident and at various recovery times. These thresholds were determined by using a Békésy-type audiometer with an interrupted tone. The ordinate is sound pressure level (SPL: db above 0.0002 dynes per square cm. in a standard 6-cc. ASA coupler).

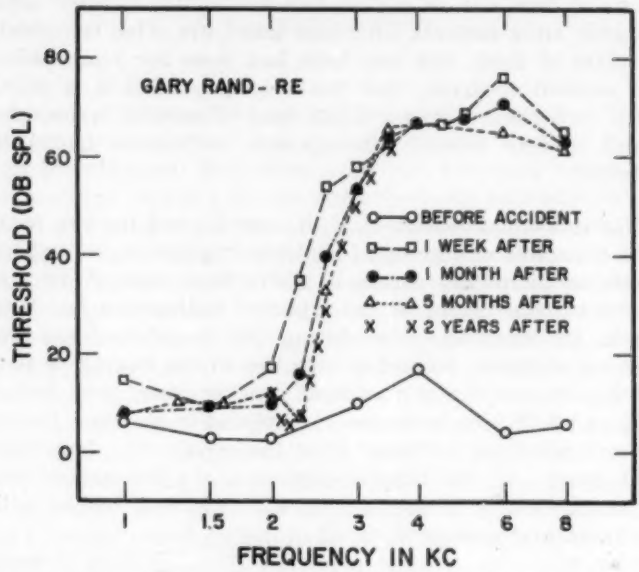

Fig. 1. Threshold sensitivity of the right ear of G. R. before and various times after explosion of a firecracker 18 inches away.

The pre-exposure thresholds correspond to hearing levels (HL) of about –10 db from 1000 to 3000 cps., 0 at 4000 cps., and –20 at 6000 and 8000 cps.; in other words, his hearing was normal up to 4000 cps., and better than normal at 6000 and 8000 cps. Since the explosion, the ear has a moderately sharp cutoff beginning at 2300 cps., with a maximum slope of about 130 db per octave.

It can be seen that after the first month, little further recovery took place. A tinnitus, loud enough to be annoying

when he becomes tired, also has persisted through the two years. Attempts to match the pitch of the tinnitus to a tone in the other ear failed because of two ostensible factors: he says the pitch is somehow "diffuse" rather than concentrated, and also fluctuates in character (timbre) in synchrony with his pulse. Probably a better match could have been made with bands of noise, but the possible information to be derived from such a match did not seem worth the effort of setting up the equipment. It is possible that the 5-db loss at 1500 and 2000 cps. can be attributed to interference by the tinnitus process.

In the five-month and two-year audiograms, a limited region of "supersensitivity" can be seen just below the cutoff frequency, *i.e.*, at 2200 cps. (it may have been present at the time the earlier audiograms were taken, but was not specifically sought by testing at closely-spaced frequencies). It is a characteristic we have often observed in permanent high-frequency losses produced by noise and gunfire. Although no complete explanation can be offered, perhaps it is due to the loss of inhibitory firing normally contributed by the fibers just basalward from the region concerned: that is, the inoperative or refractory 2500-cps. fibers.

Some tests of recruitment and diplacusis were also administered. The diplacusis tests were inconclusive, because the tone in the affected ear had such a different timbre that it could not be matched consistently to a single tone in the normal ear.

Recruitment tests were somewhat more consistent. Figure 2 shows equal-loudness contours gathered at a single session after five months of recovery. The intensity of a variable tone was adjusted by G. R. to give the same loudness as a fixed 1000-cps. tone at sensation levels of 20, 40, 50, 60, 70 and 80 db when the tones were presented alternately to the affected ear. Although there is some degree of recruitment, it is complete (at these loudness levels) only at 2400 cps.

Finally, a test for abnormal perstimulatory fatigue was also conducted. In this test, a continuous tone was presented

at 5 db SL. If the tone disappeared within one minute, the intensity was raised by 5 db; if it disappeared again, it was raised another 5 db, etc. The results were for all practical purposes negative; *i.e.*, 5-db-SL tones at 2700 and 4000 cps. were heard for a full minute, and a 6000-cps. tone had only to be raised to 10 db SL to be heard for the same time.

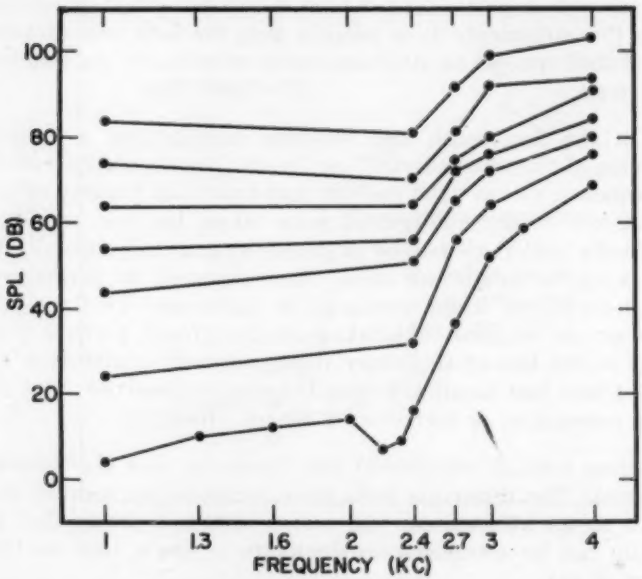

Fig. 2. Equal-loudness contours for the right ear of G. R. The bottom curve represents threshold.

The characteristics of this ear, as just described, are quite indistinguishable from those found in a study (to be published elsewhere) of ears deafened by habitual exposure to continuous steady noise or by extensive exposure to gunfire. The point is illustrated in Figure 3. The threshold of the present case is represented by the solid line. The short-dashed line is the audiogram of a 30-year-old policeman, M. L., who had never worked in high-intensity noise. Two years ago, a 38-caliber gun was discharged two feet from his left ear dur-

ing a gun battle, so the loss may be due to this incident; however, his right ear was nearly as bad, so it is possible that much of the loss shown represents a gradual accumulation of slighter traumas.

The long-dashed line shows the sensitivity of a 37-year-old man, C. R., who had worked in a steady noise of 103 db SPL

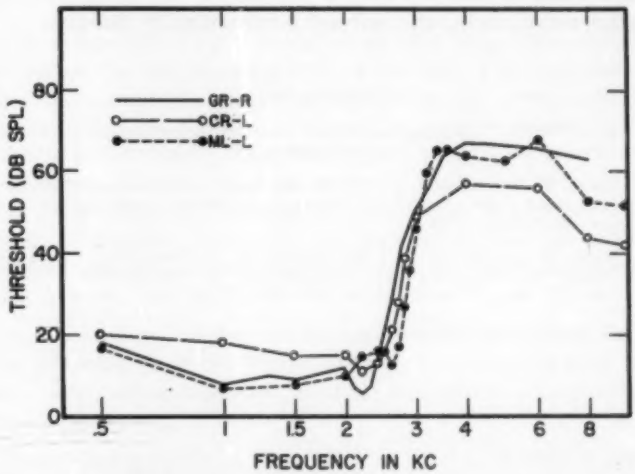

Fig. 3. Chart illustrating the similarity of hearing loss produced by different types of noise. The solid line shows an ear damaged by a firecracker, the short-dashed line denotes one probably damaged by gunfire, and the long-dashed line indicates hearing loss due to several years of exposure to steady high-intensity noise.

overall for five years. This man had no military service and had not been exposed to other types of gunfire, so his loss (which was about the same in both ears), can reasonably be attributed to his noise environment (a pre-employment audiogram taken on this ear in 1955 showed normal or better than normal at all test frequencies). The three curves are as nearly alike as would be three audiograms on the same ear taken with different typical audiometers. Clearly, then, a given high-frequency hearing loss can be caused either by prolonged exposure to steady noise or by a single incident.

SUMMARY.

This report describes some aspects of a firecracker-induced high-frequency hearing loss produced in the ear of an individual whose pre-traumatic threshold was known with certainty. A single explosion produced a permanent change in hearing level of 50 to 60 db at 3000 cps. and above. The recovery that did occur was nearly complete by one month. Other characteristics of the ear include chronic noisy tinnitus, partial recruitment, and normal perstimulatory fatigue.

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327 South Alvarado St.

HANSEL FOUNDATION COURSE IN MEDICAL OTOLARYNGOLOGY WITH PARTICULAR REFERENCE TO ALLERGY AND IMMUNITY.

This course is planned to cover other subjects in Medical Otolaryngology, such as the diagnosis and treatment of vascular headaches, Ménière's Syndrome, and related phenomena, effective use of bacterial and viral vaccines, use of antibiotics and other drugs, aerosol therapy and additional important subjects. The next course will be given May 28th through June 1st, 1962, at the Diplomat Motel in St. Louis, Missouri. Further information may be obtained by writing the Director: French K. Hansel, M.D., 634 North Grand Ave., St. Louis 3, Missouri.

BROME GRASS SEED IN PAROTID DUCT.*

FRED HARBERT, M.D.,
MAKOTO IGARASHI, M.D.,
and
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Philadelphia, Pa.

In comparison with metallic objects, vegetable foreign bodies in the air passages are uncommon. For example, of 2,042 foreign bodies requiring extraction, 155 were vegetable matter other than nuts.[1] Grasses, as foreign bodies in the bronchus and lungs, occur most often in children under 12. In a series of 35, seven were removed bronchoscopically; the rest passed through the lung and chest wall to appear on the skin.[2] Sometimes lobectomy or segmental resection of the lung is required.[5] Inhaled grass seeds can cause pneumonia, lung abscess, and death.[4]

Grass seed spikes may be driven into the tissue because of the following factors:[2] tissue squeeze, bechic blast, post tussive inspiration, and the ratchet mechanism of spikelets, glumes and awns.

The following grasses have been reported as foreign bodies[3]:

Phleum (timothy)	Cenchrus (sandbur)
Hordeum (barley)	Bromus (brome)
Avena (oat)	Alopecurus (foxtail)

Foreign bodies in salivary gland ducts are rare. A chicken feather ¾ inch long in the parotid duct of an 11-month-old infant required incision and drainage of an abscess of the parotid gland.[6] No reports of vegetable foreign bodies in salivary ducts were found in the literature of the past 16 years.

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*From the Department of Otolaryngology, Jefferson Medical College, Philadelphia, Pa.

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CASE REPORT.

H. A., a 35-year-old white male, was first seen in the Jefferson Medical College Hospital Outpatient Clinic on December 2, 1960, because of swelling and pain in the right parotid area associated with fever. About October 1, 1960, he developed a sharp pain while chewing, and he thought that his jaw was dislocated. His dentist diagnosed an abnormality of bite and dental X-rays were reported normal. On October 30, 1960, the patient first noted swelling and pain in the right parotid region. The pain subsided within two days but recurred on November 27, 1960. On November 31, parenteral penicillin therapy was given for a discharge from inside the right cheek and fever associated with swelling and pain.

Examination on December 2, 1960, showed enlargement and tenderness of the right parotid gland. Purulent material was easily expressed from the right Stenson's duct orifice and two filaments projected from the

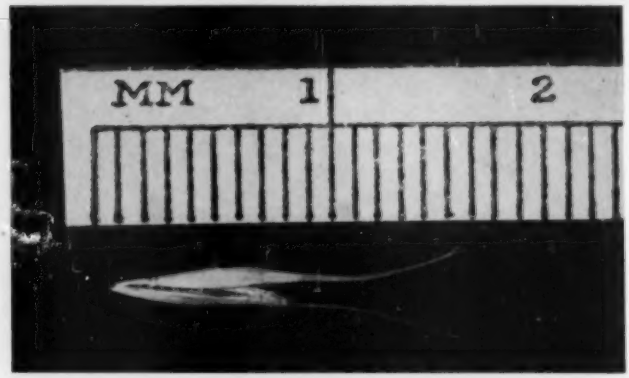

Fig. 1.

orifice. By gentle traction on these filaments a brome grass seed was removed. This was identified as a species of coarse forage grass (Bromus purgans L.) by Dr. Edgar Wherry, Professor of Botany, University of Pennsylvania. Pseudomonas aeruginosa, Streptococcus viridans and a few Neisseria species were grown from cultures of the discharge taken on December 2, 1960. Within 24 hours the symptoms and physical findings subsided. The only complaint a week later was slight excessive salivation. This patient came from the city of Philadelphia, never put grasses in his mouth, and had no idea as to the origin of the foreign body.

It is noteworthy that the only other organic foreign body in a salivary duct was a feather. This has a structure similar to the awns of grass seeds and may account for the ability of such grasses to resist the flow of saliva. Masticatory and facial movements could force the foreign body deeper in the duct; the barb-like awns would prevent its expulsion by saliva.

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THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, West Virginia, on April 23-25, 1962.

The guest speakers for Ophthalmology are:

Dr. Ramon Castroviejo, New York City, New York
Dr. Frank D. Costenbader, Washington, D. C.

The guest speakers for Otorhinolaryngology are:

Dr. Kelvin A. Kasper, Philadelphia, Pennsylvania
Dr. Fred R. Guilford, Houston, Texas

In addition, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting.

A registration fee of $25 for associate members will cover all the social and scientific sessions.

For additional information, please contact the secretary, Dr. Worthy W. McKinney, 109 East Main Street, Beckley, West Virginia.

EDITORIAL CONCEPT FOR UNRESTRICTED USE BY MEDICAL AND SCIENTIFIC PUBLICATIONS.

At Long Last!

Is there a single physician who has not been surprised and deeply concerned at some time in the middle of a dark night by the action of a drug he has given a patient? Is there a physician who has not wanted to use a drug on a critically ill patient and wished he knew a little more about the agent? Is there a practitioner who has not wished that someone would do something to systematize the deluge of information about new developments in therapy that is overwhelming us?

The practice of medicine and, even more, drug therapy, are full of surprises. As the output of pharmaceutical laboratories becomes more complex and the effects of drugs more profound in their ability to alter life's processes, the frequency of such surprises increases. Time was when a man could compound a prescription and feel quite certain of its effects; but not today.

In the past twelve months, four widely used drugs have been taken off the market because of their unexpected, injurious effect on the patient. These were not new drugs, but drugs in wide use; and now we find they are so dangerous that they must be abandoned. We believe there should be a still better, faster, and more systematic method of discovering such danger.

Some central source to which the physician could turn for complete, objective information has long been envisioned. Such a source would give the physician who is uncertain about the complete adequacy, reliability, and timeliness of his locally available drug knowledge, a great sense of security and confidence. Such a central clearing house for drug information, professionally controlled, would fill a crying need that our leaders in medicine have long expressed.

Having said this much, your editor is pleased to report

that this need is apparently to be met with a new service known as "Mediphone".

This is a system whereby physicians can obtain vital information about *any* drug *any* time, day or night, immediately, by telephoning "Mediphone" Center in Washington, D. C.

Service is available to physicians by membership. Particularly appealing to the medical profession should be the fact that "Mediphone" is conceived and operated by physicians. The service is personal and confidential and has no connection with advertising of any products. "Mediphone" receives no subsidies from private industry or the Federal Government. Calls for information will be on a physician-to-physician basis and, needless to say, will not precipitate a follow-up by a detailman.

We have also been pleased to learn that the operation of "Mediphone" has been favorably appraised by some of the country's most respected authorities on therapeutics, medical communications, and postgraduate education.

If this exciting enterprise lives up to its promise, it will fill one of the most pressing needs of every practitioner. The advent of "Mediphone" should be welcomed by all physicians.

NEW YORK EYE AND EAR INFIRMARY ALUMNI MEETING.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 9-11, 1962.

Symposia will be offered on "Complications of Stapes Surgery" and "Significance of a Mass in the Neck." There will also be a Closed Circuit Television Demonstration of surgical procedures and lectures.

Additional information may be obtained by writing to Dr. Merrill Goodman, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

The next meeting will be held January 19, 1962, at the Hotel Elysee in New York. The program follows:

1. Panel Discussion on "Problem Cases."
   (Bring Photographs of Problem Cases with you for the purpose of discussion by members of the Panel.)

2. Mandibular Deformities. Stanley L. Lane, M.D.
   Discussion by: Robin B. Rankow, M.D.
   Benito B. Rish, M.D.

Business meeting at 6 p.m., cocktails at 6:30 p.m., dinner at 7 p.m. The scientific meeting will start promptly at 8:00 p.m. Reservations, $10.00 per person for cocktails and dinner, must be sent to Samuel M. Bloom, M.D., Secretary, 123 East 83rd St., New York 28, N. Y., no later than January 15th. There will be no accommodations without advance reservations.

The Annual Spring meeting is planned for New Orleans from March 2 to March 6, 1962, including the Mardi Gras Festival. The following program has been arranged:

March 3— 9:00 a.m.—Welcoming Address—Charity Hospital Auditorium.

9:30 a.m.—Surgical Case, Dr. I. B. Goldman—Miles Amphitheater.

12:00 noon-1:00 p.m.—Lunch, Cafeteria—Tulane Medical School.

6:00 p.m.-9:00 p.m.—Cocktails, Dinner, Business Meeting—Roosevelt Hotel.

March 4—10:00 a.m.-11:00 a.m.—Topical Anesthetics, Dr. John Adriani—L.S.U. Med. School Auditorium.

11:00 a.m.-12:00 noon—Embryology of the Nose, Dr. Harold Cummins—L.S.U. Med. School Auditorium.

March 5— 9:00 a.m.-10:00 a.m.—Dressings—Miles Amphitheater, Charity Hospital.

10:00 a.m.-12:30 p.m.—Surgical Case, Dr. I. B. Goldman—Miles Amphitheater, Charity Hospital.

March 6—Mardi Gras Festival.

For reservations and further information regarding the meeting in New Orleans, please write directly to Dr. Lewis J. Rutledge, 1430 Tulane Avenue, New Orleans, La.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY, UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from April 2 through 14, 1962, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

ROYAL SOCIETY OF MEDICINE.
SECTION OF OTOLOGY.

Provisional Programme for the Session 1961/1962.

1961

November 3	Presidential Address: Mr. T. A. Clarke, M.D., F.R.C.S. (Ed.). Otitis media and other causes of deafness in children: a selective review of prevention, treatment, social and educational problems.
December 1	The tempero-mandibular joint syndrome of Costen. George Hankey, O.B.E., T.D., F.D.S., M.R.C.S.; F. C. W. Capps, F.R.C.S. Endaural meniscectomy; illustrated by a short film. A. S. Davidson, F.R.C.S. (Ed.).

1962

February 2	Vertigo of central origin. Sir Russell Brain, Bt., F.R.C.P.
March 2	Short Cases. (Details to be announced later.)
May 4	The surgery of the stapes. J. J. Shea, M.D., Memphis, Tenn., U.S.A.
June 29 (Friday)	Summer Meeting at Canterbury. (Details to be announced later.)
June 30 (Saturday)	Clinical Meeting at Kent and Canterbury Hospital.

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in the United Kingdom at the time of any of these meetings will be very welcome.

ROYAL SOCIETY OF MEDICINE.

SECTION OF LARYNGOLOGY.

Provisional Programme for the Session 1961/1962.

1961

November 3 Presidential Address: Mr. N. Asherson, F.R.C.S. Vagaries of dysphagia.

December 1 The Laryngologist and vertigo: Cough syncope. Dr. Charles Edwards, Professor Sharpey-Schafer, Sir Charles Symonds, K.B.E., C.B., and Mr. P. Golding-Wood.

1962

February 2 *The management of epistaxis.*
Management of epistaxis in familial haemorrhagic telangiectasia.....Mr. D. F. N. Harrison
Surgical management of epistaxis.
Mr. K. G. Malcomson
General medical management of epistaxis.
Mr. Chas. Joiner

March 2 *Clinical Meeting:*
Amyloid tumours of the larynx.
Dr. I. Friedman and Mr. G. Leitch
Mucor-mycosis of frontal sinus.
Dr. D. A. Osborn
Nasal "Gliomata".....Mr. James Lister

May 4 *ANNUAL MEETING:*
Centenary of Golden Square Throat Hospital.
Prof. F. C. Ormerod
Morrell Mackenzie.....Mr. R. Scott Stevenson
.....Mr. J. C. Hogg

June 29 *SUMMER MEETING* at Canterbury:
Medico-legal aspect of nose and throat surgery.
Dr. D. C. Norris and Mr. P. Addison (Secretary, Medical Defense Union).

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in the United Kingdom at the time of any of these meetings will be very welcome.

TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL POSTGRADUATE COURSE IN ALLERGY.

A continuous course of two weeks' duration is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and the Graduate School of Medicine of the University of Pennsylvania. Sessions will be held daily at the Temple University Medical Center from 9:00 a.m. to 5:00 p.m., March 5-16, 1962. Tuition Fee—$175.00. Enrollment limited. Dr. Louis Tuft is course director and Drs. George I. Blumstein and Merle M. Miller are associate directors.

The course is designed for physicians desirous of extending their knowledge of allergy. It could serve as an introductory course for those about to enter the field or as a review course for practicing allergists.

For brochure and application forms write to: Dr. George Blumstein, c/o Temple Medical Center, Philadelphia 40, Penna.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Lawrence R. Boies, Milwaukee, Wisc.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Las Vegas, Nev., November 3-9, 1962.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

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President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., Oct. 28-Nov. 1, 1962.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Alden H. Miller, 500 S. Lucas Ave., Los Angeles, Calif.
Vice-President: Dr. Herbert W. Schmidt, Mayo Clinic, Rochester, Minn.
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Treasurer: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa.
Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 1-2, 1962 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 4-5, 1962.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. John R. Lindsay, Chicago, Ill.
President-Elect: Dr. Howard P. House, 2122 W. Third St., Los Angeles 57, Calif.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 1-2-3, 1962 (mornings only).

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Howard P. House, Los Angeles, Calif.
Vice-Chairman: Dr. Joseph L. Goldman, New York, N. Y.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Paul H. Holinger, Chicago, Ill.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, O.
Meeting: Chicago, Ill., June 24-28, 1962.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Sheraton-Dallas Hotel, Dallas, Tex., April 29-30, 1962.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Charles J. Petrillo, 1441 Chapel St., New Haven, Conn.
Secretary: Dr. Robert M. Hansen, 2210 Lloyd Center, Portland 12, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Ill., Oct., 1962 (definite date to be announced later).
Annual Meeting: Belmont Hotel, Chicago, Ill., Oct., 1962 (definite date to be announced later).

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

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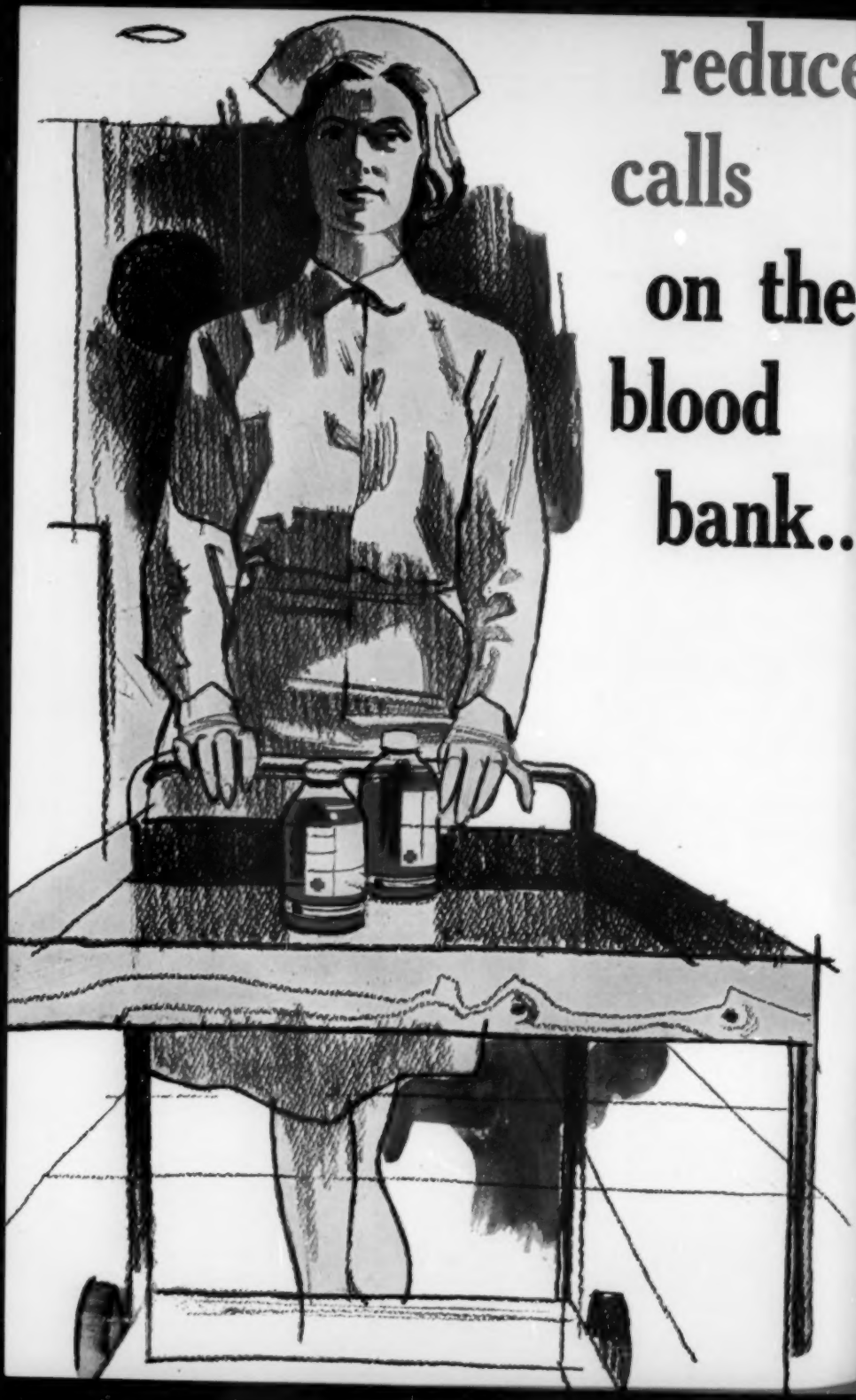

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Tablets

Adrenosem®*

Salicylate

(brand of carbazochrome salicylate)

Adrenosem helps conserve a patient's own blood. Adrenosem has become accepted pre-op medication because it minimizes the need for transfusion.

Research[1,2] has shown that lack of capillary integrity causes abnormal bleeding four times as often as do coagulation defects. Adrenosem maintains capillary integrity by decreasing excessive capillary permeability while it also promotes retraction of severed ends.

Besides reducing need for transfusion, Adrenosem's control of bleeding results in a clearer operative field . . . makes good technic even better. Adrenosem also lessens the hazard of serious postoperative bleeding . . . reduces ooze and seepage.

Adrenosem is indicated pre- and postoperatively in surgery and also nonsurgically to control bleeding associated with vascular pathosis as in peptic ulcer, telangiectasia, purpura, ecchymosis, ulcerative colitis, and others.

There are no contraindications to Adrenosem at recommended dosage levels.

supplied: For I. M. injection only—Ampuls: 5 mg. (1 cc.) and 10 mg. (2 cc.). For oral administration—Syrup: 2.5 mg./5 cc. (1 tsp.); Tablets: 1 and 2.5 mg.

references: 1. Haden, R. L., et al.: Ann. N.Y. Acad. Sc. 49:641 (May 11) 1948. 2. Cheraskin, E.: J. Am. Dent. Assn. 58:17 (April) 1959.

*U.S. Pat. Nos. 2581850; 2586294

WRITE FOR DETAILED LITERATURE
AND DOSAGE INFORMATION

THE S. E. MASSENGILL COMPANY

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storz
Surgical Instruments

N-1001 N-1003
Patent 2883980

N-1000 N-1002

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The authors concluded that "there is good reason to consider it [Terramycin] one of the most effective agents for treatment of infection of the upper respiratory tract.".

These findings confirm the continuing vitality and broad-spectrum dependability of Terramycin, as reported through more than a decade of extensive clinical use.

In brief

The dependability of Terramycin in daily practice is based on its broad range of antimicrobial effectiveness, excellent toleration, and low order of toxicity. As with other broad-spectrum antibiotics, overgrowth of nonsusceptible organisms may develop. If this occurs, discontinue the medication and institute appropriate specific therapy as indicated by susceptibility testing. Glossitis and allergic reactions are rare. Aluminum hydroxide gel may decrease antibiotic absorption and is contraindicated.

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*Jacques, A. A., and Fuchs, V. H.: J. Louisiana M. Soc. 113:200, May, 1961.

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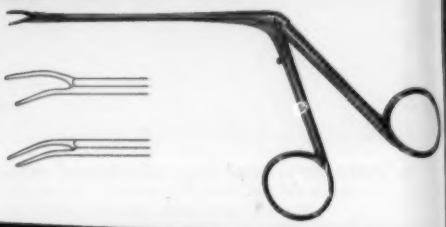

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"The 'fatigue' phenomenon, in which the nasal congestion no longer responds after frequent use of nose drops over a prolonged period, was not encountered with Tyzine solution, even in patients using it regularly for as long as two weeks."

Menger, H. C.: New York J. Med. 55:812, 1955.

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INDICATIONS: Relieves inflammatory hyperemia and edema of the nasal mucosa and congestive obstruction of sinus and eustachian ostia, as may occur in the common cold, hay fever, perennial vasomotor rhinitis, chronic hypertrophic rhinitis, and sinusitis.

DOSAGE AND ADMINISTRATION: Adults and Children 6 Years and Over – 2 to 4 drops of TYZINE (0.1%) in each nostril as needed, not more often than every three hours. When using TYZINE Nasal Spray, insert tip of plastic bottle into nostril, tilt the head slightly forward from an upright position, and squeeze sharply 3 or 4 times, not more often than every three hours.

Important: Use TYZINE Pediatric Nasal Drops (0.05%) for children under 6 years. The 0.1% concentration is contraindicated in this age group.

SIDE EFFECTS: Transient mild local irritation after instillation has been reported in rare instances.

PRECAUTIONS: Avoid doses greater or more frequent than those recommended above. Use with caution in hypertensive and hyperthyroid patients.

Overdosage may cause drowsiness, deep sleep, and, rarely, marked hypotension or even shock in infants and young children.

KEEP OUT OF REACH OF CHILDREN OF ALL AGES.

SUPPLIED: Nasal Solution, 1-oz. dropper bottles, 0.1%. Nasal Spray, 15 cc., in plastic bottles, 0.1%. Pediatric Nasal Drops, ½-oz. bottles, 0.05%, with calibrated dropper.

More detailed professional information available on request.

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Books Received

Books and monographs received are acknowledged in this column. This notice may be regarded as a return courtesy to the publisher or author. Reviews will be published later as the editors may elect.

Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

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All claims for missing journals must be made within two months of date of journal not received, otherwise we cannot guarantee complete files.

THE LARYNGOSCOPE

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